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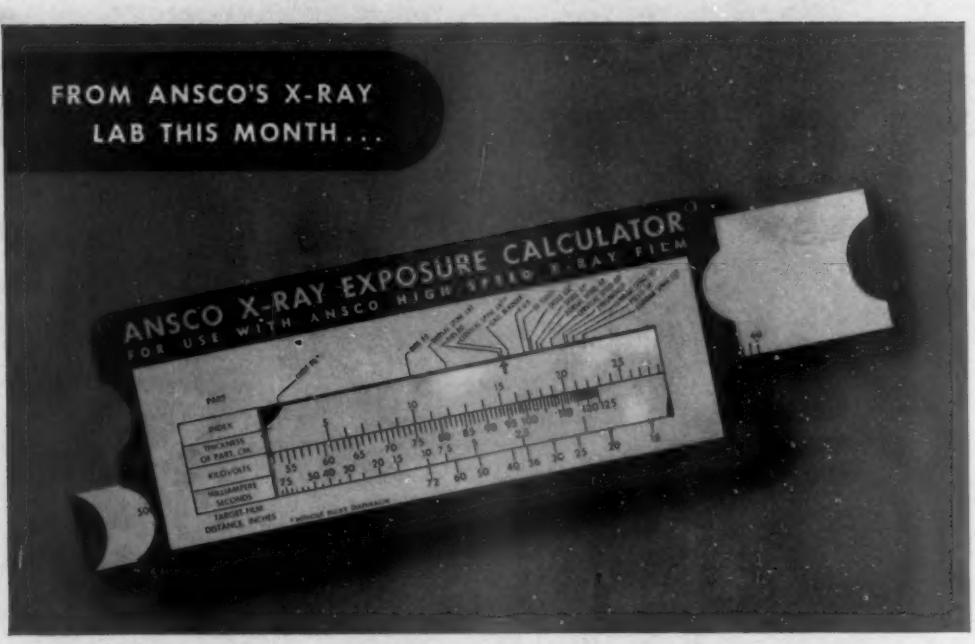

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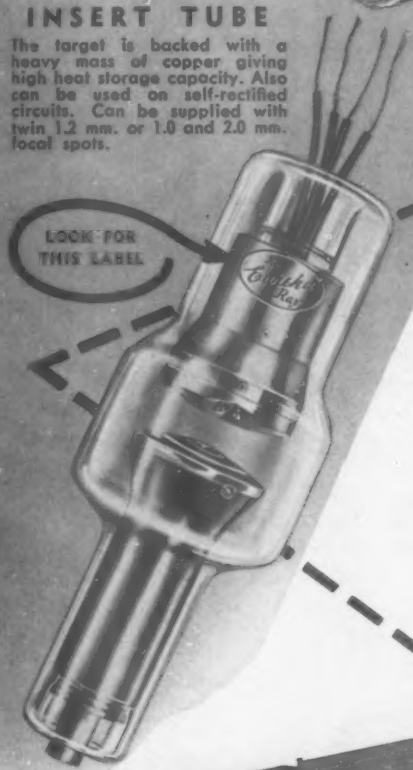

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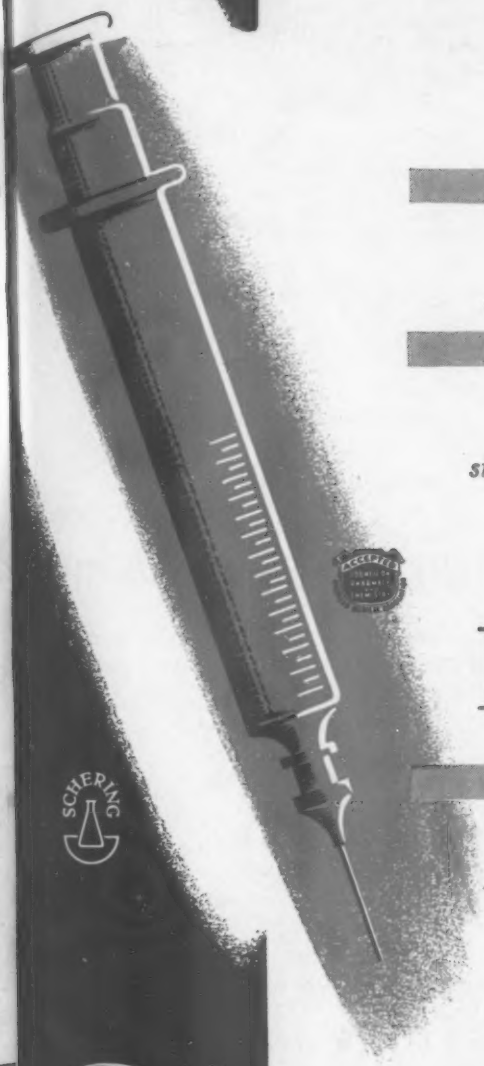

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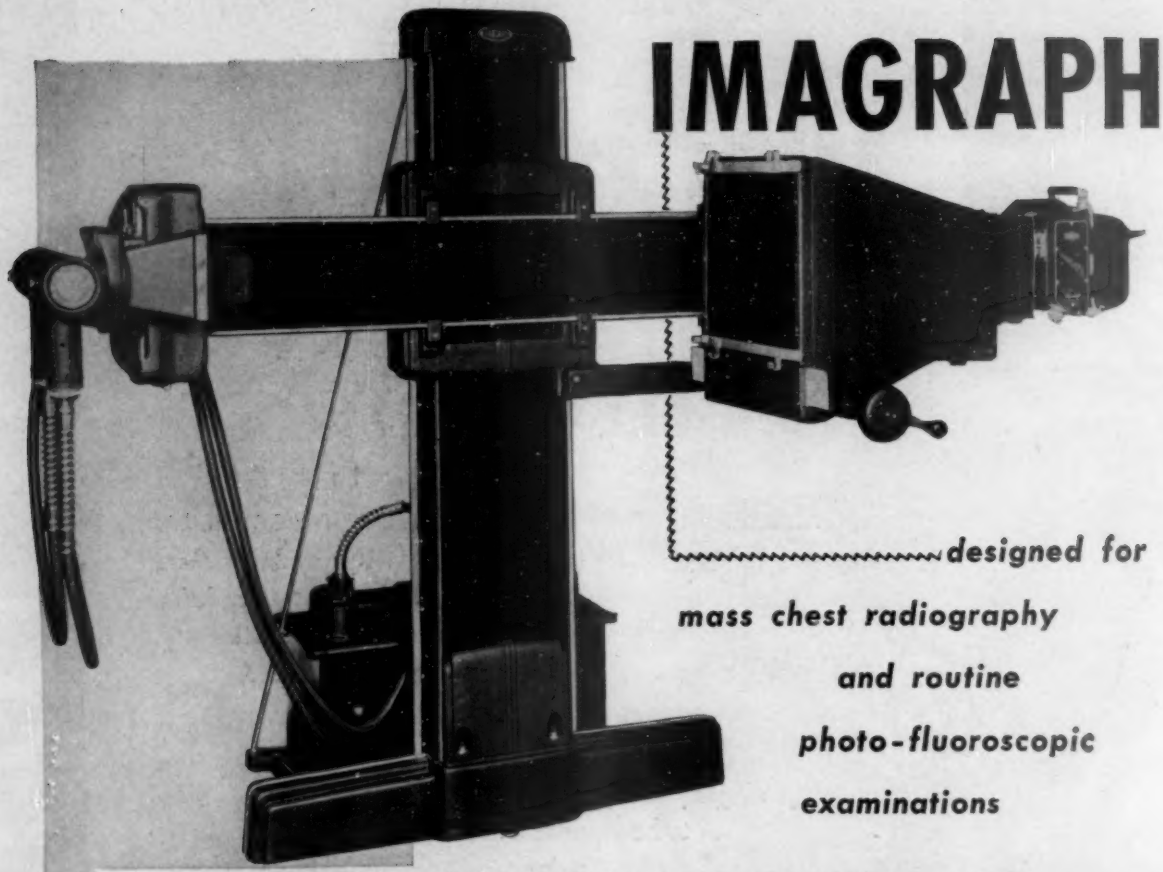

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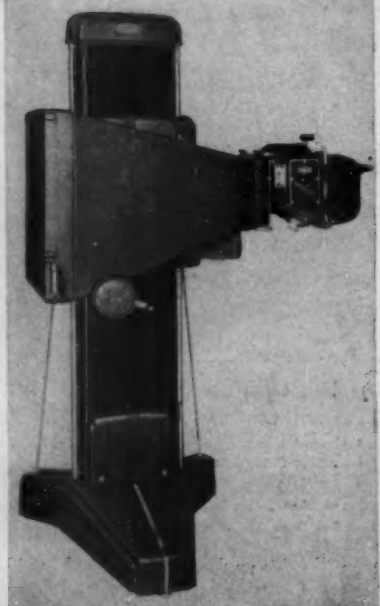

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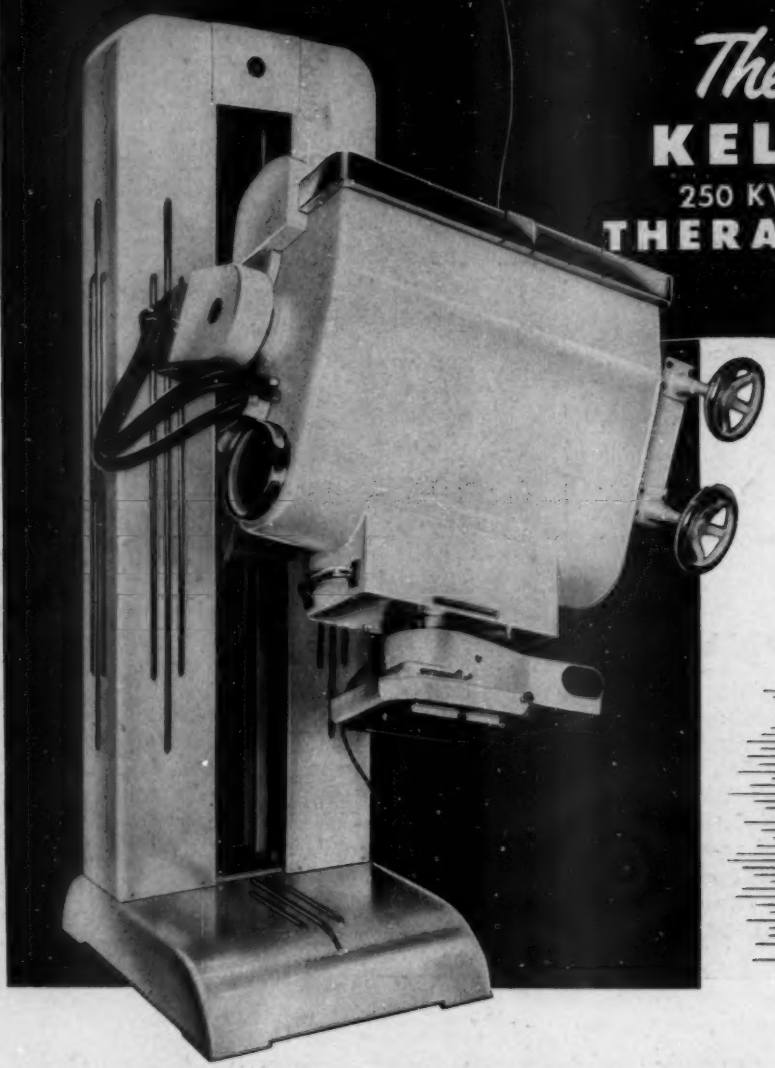

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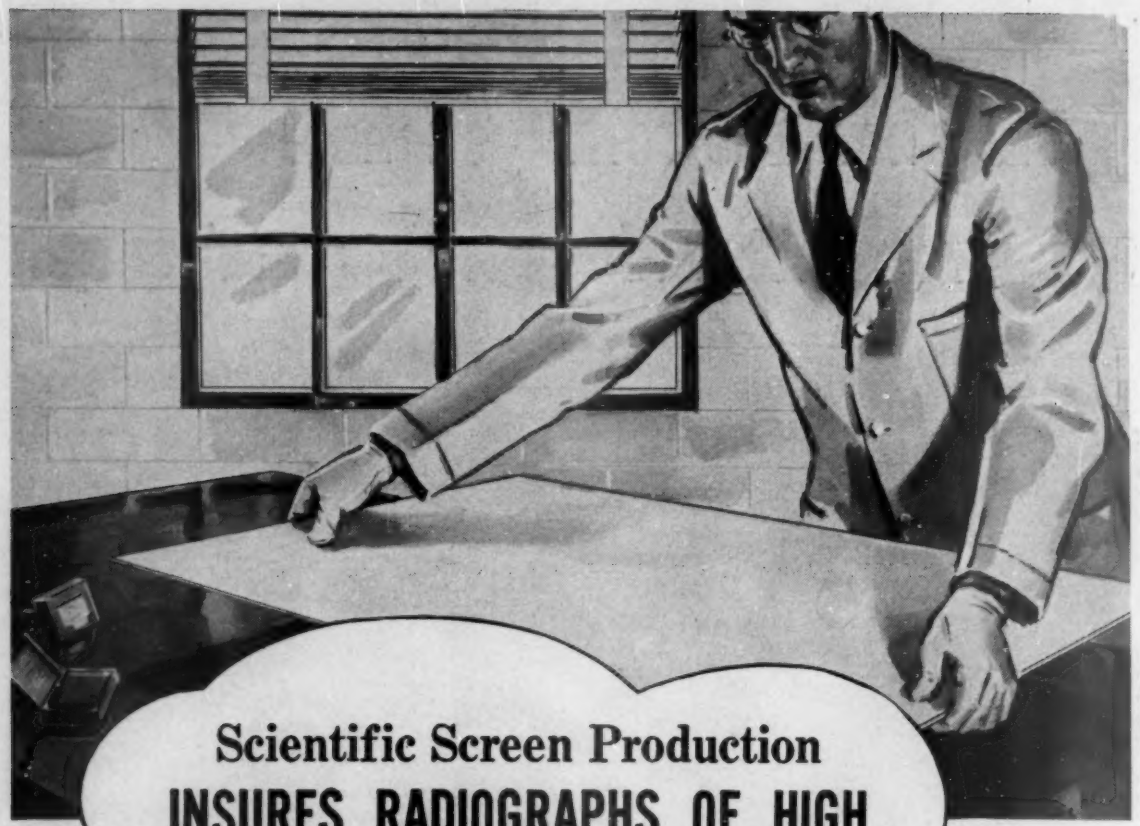

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THE CLINICAL AND ROENTGEN MANIFESTATIONS OF ERYTHROBLASTOSIS FETALIS*

By MAX RITVO, M.D., IRVING A. SHAUFFER, M.D., *and* GERALD KROSNICK, M.D.

BOSTON, MASSACHUSETTS

ERYTHROBLASTOSIS fetalis is characterized by generalized edema, jaundice, anemia, and erythroblastemia. Intrauterine death with maceration of the fetus may occur as a manifestation of this disease. The fetus may show enlargement of the liver and spleen, extramedullary erythropoiesis, and changes in the bones. The condition usually occurs in children born of mothers who are Rh negative and fathers who are Rh positive. The first-born is rarely affected unless the mother has previously received a transfusion of Rh positive blood. While Rh negative women mate with Rh positive men in approximately 10 per cent of all marriages, congenital hemolytic disease appears to occur in approximately only 1 of 250 newborns.[16] Although there is no method of combating certain fetal complications of the disease, many infants with erythroblastosis fetalis may be saved by transfusions of Rh negative blood other than that of the mother or washed maternal cells. Early recognition of the condition is of the utmost importance for the prompt institution of effective therapy. A means of determining during pregnancy whether injury to the fetus due to fetal erythroblastosis has occurred would be of inestimable value. Studies carried out at the Boston City Hospital indicate that roentgen examination of the fetus in utero affords valuable information and may permit of a definite diagnosis in certain instances.

Erythroblastosis fetalis occurs in three forms: (1) fetal hydrops; (2) icterus gravis, and (3) congenital anemia. (1) The first is characterized by accumulation of fluid in the soft tissues and body cavities, enlargement of the liver and spleen, anemia, immature red cells, and a large placenta. This type is very severe and is usually fatal during the intrauterine period or within a few hours after birth. (2) The second form, icterus gravis, is usually associated with jaundice at the time of birth or within the first twenty-four hours, although milder forms occur in which this manifestation does not develop until later, in some instances only after one to two weeks. Usually the jaundice increases in intensity and there is progressive anemia. Pulmonary hemorrhage or injury to the brain results in death. In the less severe cases, the jaundice fades in one to two weeks. With proper therapy,

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many patients in this group survive. (3) Congenital anemia is the mildest and least frequent type of erythroblastosis fetalis. The only sign in this form of the disease is extreme pallor. The condition may not be suspected and the diagnosis cannot be established unless blood studies are made. Large numbers of immature red cells are a characteristic finding. This type is self-limited.

Prior to the work of Landsteiner and Wiener, no satisfactory explanation of the syndrome which up to that time had been known as icterus gravis neonatorum was available. Darrow[3] in 1938 offered the hypothesis that it was due to an antigen-antibody reaction whose primary influence was traced to the mother, the means of transmission being the placenta. Levine and Stetson,[15] in the following year, during a study of atypical transfusion reactions, noted an irregular or atypical isoagglutination reaction in a pregnant woman who had harbored a dead fetus for a period of several months. Her serum agglutinated the husband's cell as well as those of a large percentage of group O donors. In 1940, Landsteiner and Wiener,[11] made their classical report concerning an agglutinogen in the blood of the Rhesus monkey which produced in rabbit sera agglutinins capable of reacting with a high percentage of red blood cells. This formed the basis of the theory of the Rh positive and Rh negative blood types. Wiener and Peters[28] showed that in 3 cases Rh negative persons had apparently been previously sensitized by Rh positive blood transfusions with reactions occurring during subsequent transfusions. Levine and his co-workers,[14] while studying a series of cases of repeated abortions, discovered the existence of isoagglutinins similar to the Rh factor in the blood sera. They also noted [12,13] that the mothers of erythroblastotic infants had a much higher incidence of Rh negative blood than was found in the general population. These authors indicated that erythroblastosis was due to the formation of Rh agglutinins by the mother as the result of transfusion with Rh positive blood or transmission of Rh positive fetal red cells into the maternal circulation; the agglutinins then traversed the placenta to produce agglutination and hemolysis of the fetal erythrocytes. Subsequently subgroups of the Rh type were described,[25,26,27,29] Rh′, Rh″, Rh_0, Rh_1, Rh_2, Rh_y, and Rh_x, each designating the presence of a single antigen or combination of the antigens Rh_0, Rh′ and Rh″. The occurrence of erythroblastosis in an infant with an Rh positive mother and an Rh negative father may be explained by the presence of another factor which has been termed Hr.[10,17]

Most cases of erythroblastosis fetalis are due to immunization of the mother to Rh_0. A few instances apparently result from immunization of the mother to the less common Rh′, Rh″ or one of the Hr antigens.[19] The first variety of anti-Rh antibodies to be discovered were the agglutinins. Later another type was found which had the capacity of combining with Rh positive cells but was incapable of causing agglutination when the cells were suspended in saline solution. This was termed the "blocking" antibodies type. Unger[23] believes that the agglutinins are bivalent and cause agglutination by linking of the cells, while the "blockers" are univalent. Both types act as hemolysins, no specific difference in action within the body being demonstrable.

The Rh factor is a mendelian dominant.[14] Hence, an Rh negative woman married to an Rh positive man may expect all the offspring to be Rh positive if the husband is homozygous for Rh, and half to be Rh positive if the husband is heterozygous for Rh. The pathologic condition of the fetus resulting from exposure to maternal antibodies to Rh has been variously termed erythroblastosis fetalis, congenital hemolytic anemia, hemolytic anemia of the newborn, and icterus gravis neonatorum.[19] Reactions involving anti-Rh agglutinins rarely occur unless the woman has been pregnant or has been transfused previously.[18] However, once she has developed

a concentration of antibodies sufficient to produce erythroblastosis, all subsequent offspring will be affected. The occurrence of hemolytic disease varies widely in the different series which have been reported, ranging from 2 to 5 per cent of the children born to Rh negative mothers. This indicates that Rh isoimmunization is not in every instance associated with hemolytic disease of the newborn. The male fetus is apparently more susceptible to erythroblastosis fetalis than the female.[7] Twins may not both be affected, one being Rh positive and the other Rh negative.[2,21]

The theories as to the method of production of the various forms of erythroblastosis fetalis are diverse. The anemia is believed to be the primary cause of the symptoms by some investigators.[22] Unger is of the opinion that the agglutinins cause some forms of the disease, and that the "blockers" produce other types. Darrow and Chapin[4] are of the belief that the anemia is a contributory cause, body tissue sensitization of the anaphylactic type by anti-Rh globulin being the fundamental cause of the symptoms. Similarly kernicterus is thought by Vaughan[24] to be caused by damage to the cerebral or cerebrovascular tissues by the antibody rather than being due to blood destruction.

Reports in the literature on Rh isoimmunization in pregnancy indicate that the diagnosis of impending development of hemolytic disease in the infant may be made with moderate certainty by titer estimations. Also, the severity of the manifestations may be estimated in a general manner by the titer in the maternal blood. Prior to actual death of the fetus, objective evidence of Rh incompatibility has not been available. Primrose, *et al.*[20] have grouped titer variations with blocking and agglutinating antibodies and the sequences of the changes in the titers serve as a means of indicating probable injury to the fetus. However, the degree of titer dilutions has in many instances not corresponded to the severity of the disease.

The Rh factor is an agglutinogen which is found in erythrocytes of approximately 85 per cent of Caucasians irrespective of sex or primary blood groups. The white race has the greatest incidence and several groups are composed entirely of Rh positive individuals. Incidences of 24.6 per cent in Belgians and 33.6 per cent in Basques indicate that these are the highest Rh negative groups. Rh negative types apparently do not occur in Indians, Filipinos, Japanese, Chinese, Negroes, and certain other groups.

The mortality in erythroblastosis fetalis has been very high, ranging from 80 to 100 per cent. With earlier and more definite diagnosis and prompt therapy, there has been a marked improvement in this respect. In a series of cases reported by Cole,[1] the deaths decreased from 85 per cent in 1933–1936 to 42 per cent in 1946. There were no survivors in the hydropic form of the disease; the group with jaundice had a mortality of 43 per cent; the anemic type showed 77 per cent survivals.

The blood studies in patients suspected of harboring an erythroblastotic fetus should comprise typing of both parents and anti-Rh titer estimations in the mother. Because of the occurrence of blocking antibodies, it appears advisable that tests for "blocking" should also be performed. The absence of Rh antibodies usually means that the infant will be normal, while the presence of Rh antibodies in the mother during the pregnancy indicates the probable occurrence of erythroblastosis fetalis. Cases in which the mother is Rh negative, particularly if the history indicates that previous pregnancies had been complicated by erythroblastosis fetalis, should be investigated to determine whether the father is homozygous or heterozygous for the Rh factor. During pregnancy antibody titers should be recorded at frequent intervals after the seventh month of pregnancy. As noted above, the level of the titer is not an entirely dependable index of the degree of the condition, although Overstreet, *et al.*[16] and Howard, *et al.*[8] state that the duration of the presence of the anti-Rh titer in the

mother serves in a general way to indicate the severity of the hemolytic disease. It appears that determination of whether injury to the fetus has taken place is best made by blood studies at approximately ten weeks prior to term. If an Rh negative mother first presents antibody titer less than ten weeks antepartum, spontaneous labor is probably advisable. If the antibody titer is first observed during the tenth to fourteenth week antepartum, early induction of labor should be seriously considered, particularly if clinical and roentgen observations do not demonstrate evidence of fetal death. On the other hand, the presence of antibody titer fourteen or more weeks prior to term is strongly suggestive of serious injury to the fetus. In these cases, induction of labor is usually contraindicated as the possibility of viability is extremely remote, the hazards to the infant being so great because of prematurity. Clinically, there is no means of determining definitely whether termination of the pregnancy in a suspected case of erythroblastosis fetalis is indicated. In the past, the high fetal mortality attendant on early induction of labor indicates that it is not, as a rule, a justifiable mode of treatment.

The roentgen findings in the cases of erythroblastosis fetalis observed by us may be divided into three main groups: (1) soft tissue changes consisting of generalized edema and enlargement of the liver and spleen; (2) abnormalities of the skeleton, and (3) evidences of fetal death.

(1) *Soft Tissue Changes.* There occurs massive edema with very marked swelling and thickening of the soft tissues of the head, face, and chin, producing a halo-like appearance about the head, face and neck. The edematous fleshy tissues of the body and limbs appear thicker and denser than normal on the roentgenogram. The changes in the soft tissues may be obscured in utero by the amniotic fluid and the overlying densities of the maternal structures.[9] However, in many instances the fetus is well visualized and the above described changes may be demonstrated with a surprising degree of clarity. The enlargements of the liver and spleen are not seen in utero as the density of the internal organs is uniform during fetal life, aeration of the lungs and the presence of gas in the gastrointestinal tract being necessary to outline the shadows of the diaphragm, liver, and spleen.

(2) *Skeletal Abnormalities.* The osseous changes in erythroblastosis fetalis were first described by Follis, Jackson and Carnes[6] and consist of a marked increase in the density of the bones. The skull, vertebrae, ribs, pelvis, and long bones may be affected in varying degrees. In the skull the sphenoid and occiput are principally involved, the remainder of the calvaria showing less or no involvement. The vertebrae show marked sclerosis. In the ribs, the density is most clearly visualized at the angles. The pelvis and other flat bones may be diffusely eburnated. The long bones in the cases observed by us revealed uniformly increased density throughout the entire shafts with marked narrowing or obliteration of the medullary spaces. Follis, Jackson and Carnes described zones of diminished density of varying width at the ends of the tubular bones immediately below the cartilage shaft junctions. On carefully executed roentgenograms, the abnormalities in the bones may be demonstrable in utero. This may be of great value in diagnosis as it enables the clinician to determine during the course of the pregnancy whether the fetus is erythroblastotic or normal.

The change in the density of the bones is demonstrable both on the roentgenogram and microscopically. There is an actual increase in the number and thickness of the bone trabeculae. This is apparently due to lack of destruction of the calcified cartilaginous matrix substance which is then covered with a thick layer of bone. The cause of these changes is not clear. Various theories have been suggested. Day and Follis[5] report increased density at the ends of the growing bones of young animals who had received large amounts of estrogenic hormone. The placenta in eryth-

roblastosis fetalis is very large and apparently contains increased amounts of chorionic hormone. In utero, blood formation also occurs in extramedullary tissues and the hyperplastic marrow may affect the bones of the fetus differently than after birth. It may be of importance to note in this connection that in congenital lues there is increased density of the bones. Also in osteopetrosis (Albers-Schönberg disease), known as osteosclerosis fragilis generalisata, there are bone changes very similar to those in erythroblastosis fetalis. However, multiple fractures are common in osteopetrosis but have not been noted in erythroblastosis.

(3) *Evidences of Fetal Death.* Death of the fetus occurs with resultant stillbirth in many cases of erythroblastosis fetalis. Overlapping of the bones of the skull is the earliest and most reliable sign that the fetus has died. During active labor, the bones of the fetal skull may undergo varying degrees of compression and overlap slightly at the sutures. Except when the patient is in labor, however, overlapping of the skull bones is considered pathognomonic of fetal death. This change may occur within a few days or weeks after the cessation of life. Sharp angulation of the head in relation to the spine, lordosis of the caudal extremity of the spinal column, collapse of the thoracic cage, and marked, generalized decalcification of the fetal skeleton are also accepted as roentgen manifestations of death of the fetus; these are later in developing and are not always dependable criteria, however. Marked disproportion between the size of the fetus and its expected development at the supposed clinical duration of the pregnancy is significant in this respect.

The demonstration of the above-described changes in utero is difficult. The utmost care is essential in the production of as nearly technically perfect roentgenograms as possible. High milliamperage technique is necessary. A rotating anode tube of 200 ma. capacity has produced satisfactory results in our clinic. The Potter-Bucky diaphragm, a compression band applied over the abdomen, and a small cone are required. Rapid exposure is essential to eliminate the possibility of fetal movement during the making of the roentgenogram. The patient must be cooperative and suspend movement and respiration completely during the roentgen exposure. Anteroposterior, oblique, and lateral projections are made of each patient. The edema of the fetal tissues and the hydramnios which are so often present in this condition are very apt to obscure the alterations in the densities of the bones and soft tissues of the fetus or the roentgen manifestations of fetal death. Frequent re-examination may be necessary to detect early changes. The first roentgenograms should be made approximately ten to fourteen weeks prior to term and thereafter at intervals of three to six weeks. A sudden change in titer, clinical evidence of fetal death, or signs of toxemia are important indications for careful roentgen studies of the fetus. As with other rare and unusual conditions, the diagnosis will be made only if the manifestations are clearly understood, constantly borne in mind, and painstakingly sought for in every instance. The importance of early diagnosis in the planning of therapy and making a proper prognosis is self-evident.

CASE REPORTS

Case 1. M. H., white, aged forty. The patient had had nine previous pregnancies. The first seven pregnancies resulted in full-term, normal deliveries. In the eighth pregnancy, artificial labor was induced three weeks before term because of eclampsia with hypertension. The ninth pregnancy terminated in a stillbirth at term, apparently due to hypertension. The present pregnancy progressed uneventfully during the first eight months. The mother weighed 160 pounds and the blood pressure was 124/82. She was found to be Rh negative, blood group O. The Hinton test was negative. Three days before delivery she entered the hospital with severe headaches and vomiting. She had felt no fetal life for one week and the fetal heart sounds were not heard. There was

FIG. 1*A*. Case 1. Three weeks prior to delivery. The fetal skeleton is well outlined. There is no roentgen evidence of fetal death.

FIG. 1*B*. Case 1. Anteroposterior projection. Two days prior to delivery. There is overlapping of the occipitoparietal suture indicating fetal death. The fetal skeleton is poorly visualized because of marked hydramnios.

FIG. 1*C*. Same as Figure 1*B*, oblique view. There is increased density in the bones of the skull, particularly in the region of the base, at the angles of the ribs, and the long bones. The edema of the scalp produces the typical "halo."

FIG. 1*D*. Case 1. The fetal skull, after delivery. The overlapping of the bones at the occipitoparietal suture and the markedly increased density of the bones of the skull are well illustrated.

marked hydramnios. Titer was 1:16 in albumin; negative in saline. It was believed that there was fetal death at term, probably associated with erythroblastosis. Roentgen study three weeks previously had revealed a single fetus

Fig. 1*E*. Case 1. Soft tissue roentgen studies of the head show the marked edema of the scalp.

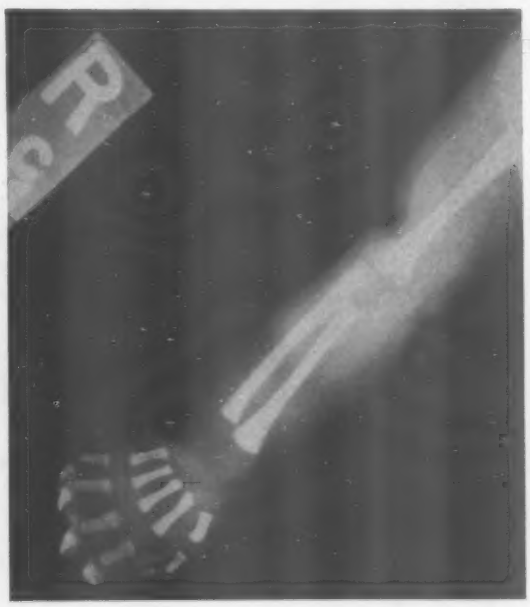


Fig. 1*F*. Case 1. The bones of the arm and hand show marked sclerosis with cortical thickening and obliteration of the medullary cavities. There is generalized edema of the soft tissues.

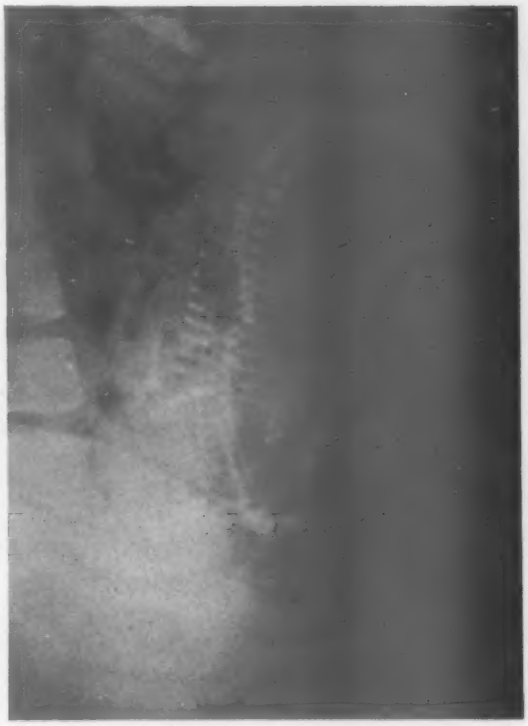
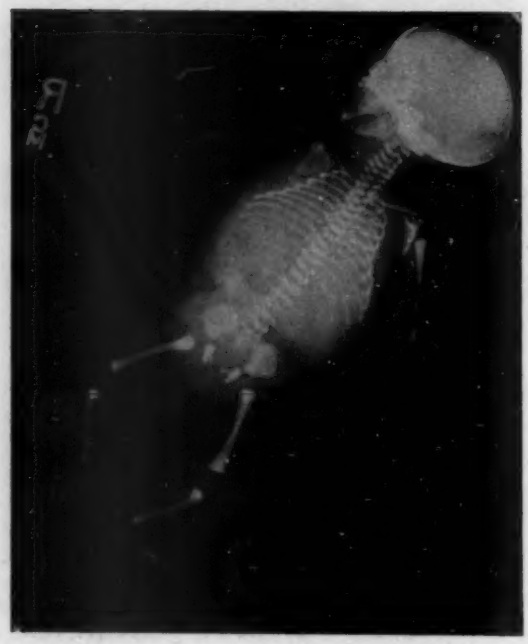


Fig. 2*A*. Case 11. Eleven days prior to delivery. There is extensive sclerosis involving practically the entire skeleton.

Fig. 2*B*. Case 11. Stillborn fetus. The sclerotic changes in the bones and the generalized edema are well illustrated.

FIG. 2C. Case II. The bones of the base, the occipital bone, the facial bones, and the cervical vertebrae are extremely dense. There is overlapping of the bones of the skull. There is very marked edema of the scalp and chin.

FIG. 2D. Case II. Anteroposterior view of the skull, illustrating the changes associated with erythroblastosis fetalis.



and at this examination there was no roentgen evidence of fetal death. Nineteen days later, the roentgenograms showed definite overlapping of the fetal skull bones consistent with death of the fetus. The bones of the base of the fetal skull, the long bones and the ribs were poorly visualized but showed increased density. Three days subsequently, the patient was delivered of a 10 lb. 8 oz., stillborn, macerated female infant.

CASE II. A. D., white, aged thirty. There were two previous full-term, normal pregnancies. The patient was Rh negative, blood type

FIG. 3A. Case III. Stilborn fetus. There is increased density involving the bones of practically the entire skeleton. The lungs are non-aerated and no gas shadows are present in the gastrointestinal tract. The liver and spleen are markedly enlarged. There is edema of the scalp and the extremities.

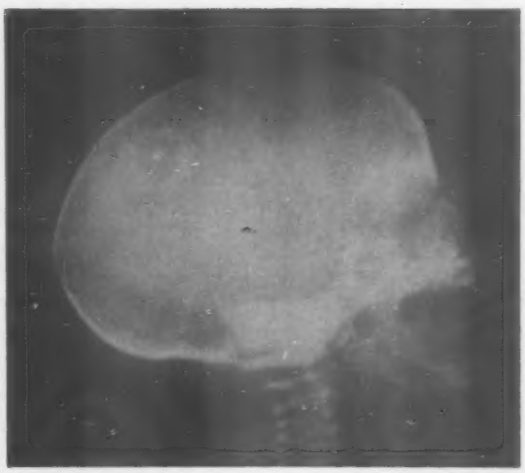


FIG. 3*B*. Case III. Low kilovoltage roentgen study of the head demonstrates the marked edema of the scalp and neck.

FIG. 3*C*. Case III. There are sclerotic changes in the bones of the pelvis and lower extremities with edema of the soft tissues.

FIG. 4*A*. Case IV.

FIG. 4*B*. Case IV. The bones show moderate sclerotic changes. There is marked edema of the head, face, chin, and extremities. Small amounts of gas are present in the stomach, duodenum, and proximal jejunal loops.

O, and her husband was Rh positive. The last menstrual period had been on February 7, 1947. On September 3, 1947, the titer was 1:64 in albumin and 1:2 in saline; on September 29, 1947, the titer was 1:256 in albumin and 1:4 in saline. About four weeks later, October 26, 1947, she spontaneously delivered a fetus which was stillborn and macerated.

Case III. H. M., white, aged twenty-one. Five years ago, she had a full-term, normal delivery. A year later, there was a miscarriage at three and a half months. One year subsequently a child born at term developed jaundice six days after birth. She was Rh negative and the husband was Rh positive. The titer estimations during the pregnancy were as follows: September 20, 1947, 1:32 in albumin and negative in saline; September 26, 1947, 1:128 in albumin, negative in saline; October 10, 1947, 1:128 in albumin and negative in saline. On October 26, 1947, she spontaneously delivered a stillborn, markedly macerated fetus.

Case IV. M. V. S., white, aged twenty-two. She had had three previous pregnancies which had resulted in full-term, normal deliveries. Two years ago, a fourth pregnancy terminated in a stillbirth due to erythroblastosis, the fetus being macerated and icteric. In the present pregnancy, the titer estimation during the sixth month was 1:8 in albumin and negative in saline. Two weeks later, the titer was 1:32 in albumin and negative in saline. On April 3, 1948, about a week prior to the expected date of delivery, she noted a marked increase in the size of the abdomen and had felt no fetal activity for several days. No fetal heart sounds could be detected. Titer estimation was 1:256 in albumin and negative in saline solution. Spontaneous labor resulted in the delivery of a full-term infant, with marked edema, ascites, hepatomegaly, and splenomegaly, which died in approximately forty-five minutes. The placenta was large, pink in color, and edematous. The autopsy revealed erythroblastosis fetalis of the hydropic type with ascites, anasarca, bilateral hydrothorax and atelectasis, and marked enlargement of the liver and spleen.

SUMMARY AND CONCLUSIONS

In erythroblastosis fetalis there is generalized edema, jaundice, anemia, and erythroblastemia. Intrauterine death and maceration of the fetus may occur with enlargement of the liver and spleen, extramedullary erythropoiesis, and changes in the bones.

The condition is usually seen in children born of mothers who are Rh negative and fathers who are Rh positive.

Titer estimations in the maternal blood indicate the impending development of hemolytic disease of the infant and may serve as an index of the severity of the condition. Determination of injury to the fetus is best made in the last weeks of the pregnancy.

Studies carried out at the Boston City Hospital indicate that roentgen studies of the fetus in utero afford valuable data and may permit of a diagnosis of erythroblastosis fetalis in certain instances.

The roentgen findings in erythroblastosis fetalis comprise (1) soft tissue changes consisting of generalized edema and enlargement of the liver and spleen; (2) abnormalities of the skeleton with increased densities in the bones, and (3) evidences of fetal death.

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PRIMARY HEMANGIOMA OF THE SKULL: A RARE CRANIAL TUMOR

REVIEW OF THE LITERATURE AND REPORT OF A CASE, WITH SPECIAL REFERENCE TO THE ROENTGENOGRAPHIC APPEARANCES

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1. INTRODUCTION

IN GENERAL, hemangiomata are rare skeletal tumors. In a series of 1,831 bone tumors recorded at the Registry of Bone Sarcoma, only 13 examples of hemangioma of bone occur. Christensen's (1925) series of 918 bone neoplasms contained 11 hemangiomata, while Geschickter and Copeland (1932) noted only 12 examples in a series of 1,700 neoplasms of bone. Thus in a total series of 4,449 neoplasms of bone, only 36 (or 0.7 per cent) are hemangiomata.

Occurring in the skull, hemagiomata are even more unusual. In the Bone Registry series, only 3 cranial angiomata are noted, while Geschickter and Copeland recorded 5 examples among their cases. Töpfer (1928) found 3 cases of angioma of the skull in 2,154 consecutive bodies examined at postmortem. Summing the first two series, it is apparent that in a total of 3,531 neoplasms of bone, only 8 angiomata of the skull are recorded, an incidence of 0.2 per cent.

Although cranial hemangiomata are rare, the skull constitutes the commonest site for such lesions, apart from the vertebral column in which the majority occur. Töpfer found vertebral angiomata in 257 of the 2,154 bodies he examined, and Bucy and Capp (1930) stated that hemangiomata are found more commonly in the vertebral column than in all the other bones of the body put together. In the extravertebral cases, the skull is more frequently involved than the other bones of the skeleton. In a series of 33 hemangiomata of bones other than the vertebrae, Bucy and Capp found the skull to be the site of the lesion on 18 occasions. Geschickter (1936) found 5 angiomata in a series of 39 primary benign tumors of the skull. Thus hemangiomata of bone most commonly are tumors of the axial, rather than the appendicular skeleton.

It is therefore apparent that primary hemangioma of the skull is a most unusual tumor. For that reason the present paper describes a case of such a neoplasm in which a preoperative diagnosis was made, with special emphasis on the roentgenographic features, which constitute the diagnostic *sine qua non*.

2. ANALYTICAL REVIEW OF LITERATURE

For obvious reasons, case reports of primary hemangioma of the skull are relatively infrequent in the literature. Moreover, complicating factors are introduced, especially in the older records. Some of these tumors have been classed under the old term of "melanotic sarcoma" (e.g. Cushing, 1923) and hence have not been recorded as hemangiomata. Other examples have been described, which are not primary tumors of the skull, but which form one of a number of such lesions scattered through the body. Some of these cases have been examples of Lindau's disease, or other unusual syndromes, while others have been the result of extension of hemangiomatous lesions from the brain into the skull. Still others have been listed as "aneurysm of bone."

In an endeavor to clarify the historical background, the literature of the last one hundred years has been carefully searched, and in Table 1 are listed all the cases of

cranial hemangiomata, primary or otherwise, which were found.

The first case of hemangioma of the skull was described about a century ago by Toynbee (1845, 1847), and since then a further 59 examples have been added. There thus exist in the literature reports of 60 cases in which angiomatous lesions have been found in the skull. The author's case brings the total to 61. The details of these case reports are summarized in Table 1.

However, not all of these cases are examples of primary hemangioma of the skull. In 10 of the reports, hemangiomatous lesions are described in other parts of the body, especially the liver; at least 3 of these 10 cases are examples of Lindau's disease. In a further 2 cases the lesion does not arise primarily within the cranial bones, but involves them secondarily. In 9 of the reports the descriptions are too cursory to provide adequate data for detailed analysis. Therefore, there remains a group of 39 definite examples of primary hemangioma of the skull, which, with the author's case included, is increased to 40.

This group of 40 verified primary hemangiomata of the skull forms the basis for the subsequent analyses and statistical data. The relevant details from each of the case reports are found in Table 1.

(*a*) *General Incidence.* The general incidence of primary hemangioma of the skull has already been discussed. These tumors

TABLE I

SUMMARY OF RECORDED CASES OF CRANIAL HEMANGIOMATA

Author	Year	No. of Cases	Age	Sex	Site in Skull	Result	Other Lesions
Toynbee	1845	1	19	M	Biparietal	Died	Tuberculosis
Ehrmann	1847	1	40	F	Left parietal	Excised	Postoperative meningitis
von Rokitansky	1856	1			Right parietal		—
Cruveilhier	1856	1	38	F	Multiple (12) in skull	Died	Also in femur, 3rd rib, shoulders
Bilroth	1856	1			Multiple in skull	Died	Also in face, spleen and liver
Sengalli	1860	1		M	Occipital	Died	Pulmonary disease
Virchow	1867	1	72	F	Right parietal	Died	Also in liver
Morris	1880	1	40	F	Right parietal	Punctured	—
Stamm	1891	1	4/12	F	Multiple in skull	Died	Also in brain and other bones and organs
Pilcher	1894	1	15	F	Frontal	Removed piecemeal	
Zajaczkowski	1901	1	38	F	Left parietal	Removed	—
Ziegler	1901	1			Vault of skull (parietal)		—

TABLE I (*continued*)

Author	Year	No. of Cases	Age	Sex	Site in Skull	Result	Other Lesions
Politzer	1901	1	12	F	Right petrous	Died	—
Schöne	1905	1	39	M	Left occipital	Removed	—
Planchu	1911	1					
Major and Black	1918	1	34	M	Left frontal and right temporal	Died	Also in liver, cystic adrenals
Brandt	1921	1	47	M	Left petrous	Died	Also in right retina, left cerebellum, kidneys, etc.
Kaufmann	1922	1	76	M	Vault of skull	Died	Also in liver and other organs
Cushing	1923	1	55	F	Left parietal	Removed	—
Christensen	1925	1	55	F	Left parietal		—
Lanari and Marque	1925	2			Frontal Frontal	Treated with roentgen rays	Extensive nevi of face and scalp with hemiplegia and epileptic seizures
Bregman and Mesz	1927	1					
Dandy	1928	2			Frontoparietal Occipital		Arose primarily from dura, not diploe
Erös	1928	1	70	F	Right and left frontal	Died	
Töpfer	1928	3					
Eigler	1930	1	62	M	Multiple in skull	Died	—
Geschickter	1936	3	17/12	F	Congenital in skull	Removed	—
Dikansky	1932	2	20 17	M F	Left temporal and right parietal Right parietal	Removed Removed	—
Reischauer	1933	1	40	F	Biparietal	Died	—
Overend	1933	1	28	M	Occipital	Removed	—
Sterling and Joz	1935	1					
Mazzini and Brachetto-Brian	1935	1	39	F	Right frontal	Removed	—

Table I (*continued*)

Author	Year	No. of Cases	Age	Sex	Site in Skull	Result	Other Lesions
Abbott	1937	1	3	F	Right frontal	Removed	—
Anspach	1937	1	11	F	Left parietal	Unsuccessful attempt at removal	—
Porta and Clivio	1938	1	49	F	Right parietal	Removed	—
Pich	1938	1	44	M	Left parietal	Removed	—
Sommer	1938	1	16	F	Occipital	Removed	—
McKenzie	1939	1	34	F	Right petrous	Deep roentgen therapy	
Kaplan and Kanzer	1939	1	47	F	Left parietal	Removed	—
Irgens	1939	1	28	F	Right occipital and right petrous	Treated with radium. Died 9/12 later	—
Mangabeira-Albernaz	1939	1	21	F	Middle ear, petrous and temporal bone	Died	—
Vincent and Brégeat	1939	1			Right basisphenoid	Died	Trigeminal neuralgia
Abbott	1941	1	3/52	M	Right parietal	Removed	—
Janker	1941	1			Frontal	Treated with radium	Also in face
Lange and Taques-Bittencourt	1941	1			Right frontoparietal		—
Pettit-Dutaillis, Bertrand and Messimy	1941	1					Hemangioendothelioma
Echols and Kleinsasser	1942	1					
Rowbotham	1942	5	32 49 66 37 ?	F F F F ?	Right frontal Left frontal Right orbital roof Left frontal Parietal	Removed Removed Removed Removed	— — — — —
Paul	1946	1			Orbital	Removed	—
Wyke	1946	1	28	F	Left frontal	Removed	—

constitute about 0.2 per cent of all osseous neoplasms, and about 10 per cent of the primary benign tumors of the skull, which, in themselves, are rare.

(*b*) *Age Incidence.* In the series of 40 cases under discussion the oldest patient was aged seventy years (Erös, 1928), and the youngest three weeks (Abbott, 1941). The decadal distribution of the primary hemangiomata of the skull is represented in Table II. From this it is apparent that

TABLE II

THE DISTRIBUTION BY DECADES OF 40 CASES OF PRIMARY HEMANGIOMA OF THE SKULL

Decade	Years	Number of Cases	Per-centage
1	0–10	3	7.5
2	11–20	7	17.5
3	21–30	4	10.0
4	31–40	9	22.5
5	41–50	4	10.0
6	51–60	2	5.0
7	61–70	3	7.5

these tumors have their highest incidence (22.5 per cent) in the fourth decade and are somewhat less frequent in the second decade (17.5 per cent). They are most infrequent in the sixth decade (5.0 per cent). The average age for the group is 29.8 years. Only 14 cases of the series are aged less than thirty years. These findings are in accord with those of Gross (1879) who found hemangioma of bone to be rare before the thirtieth year.

(*c*) *Sex Incidence.* Primary hemangioma of the skull is three times as frequent in females as in males. The sex of the patient is noted in 33 of the 40 cases listed, and in 25 of these the patient was a female, while only 8 were males. There is no correlation between age and sex.

(*d*) *Site in Skull.* Hemangiomata have been described in practically all the bones of the skull, but the primary hemangiomata seem to have a special predilection for the parietal bones. The distribution of the 40 examples of primary hemangiomata in the different parts of the skull is seen in Table III.

In 7 cases more than one lesion was present in the skull. Counting these multiple lesions separately, it is apparent that the

TABLE III

LOCATION OF PRIMARY HEMANGIOMATA OF THE SKULL IN THE CRANIAL BONES

Site in Skull	Number of Lesions	Per-centage
Orbital	2	4.5
Frontal	10	22.9
Parietal	20	45.6
Occipital	6	13.6
Petrotemporal	5	11.4
Basisphenoid	1	2.3

parietal region was the seat of the tumor on 20 occasions, while the frontal bones were only involved 10 times. Thus of the individually identifiable neoplasms, just under half (45.5 per cent) were located in the parietal bones.

Toynbee's original case showed a tumor in each parietal region, and he suggested that the lesion was related to the centers of ossification of the bones. This suggestion has been repeated (Erös, 1928; Reischauer, 1933), but there is no statistically significant relation to the ossific centers of the cranial bones, when the series is reviewed as a whole.

(*e*) *Etiology.* As yet there is nothing definite that can be said on this question. Some cases may be of congenital origin, and there are at least 3 such in the literature (Geschickter, 1936; Abbott, 1937; Abbott,

1941). Some authors have suggested that all primary hemangiomata of the skull are of congenital origin, but this can hardly be true in view of the age distribution of the recorded cases.

In a significant number of cases of primary hemangioma of bone the history dates from some local trauma. A history of trauma may often be elicited in cranial examples of the neoplasm (e.g. Pilcher, 1894; Dikansky, 1932: Mazzini and Brachetto-Brian, 1935; Kaplan and Kanzer, 1939; Rowbotham, 1942), as in the case reported in the present paper. Whether this is of definite etiological significance, or merely brings to fruition a hitherto quiescent neoplasm, remains to be decided.

3. CLINICAL DIAGNOSIS

It is not possible to diagnose a primary hemangioma of the skull by purely clinical methods.

The history is not specific. There is usually a story of a hard lump on the head which has been present for months, or even years. Hemangiomata of other bones of the skeleton may present with an acute history, perhaps related to trauma (Bucy and Capp, 1930). But in the majority of the extracranial lesions, especially those in the vertebrae (Makrycostas, 1927; Töpfer, 1928) there may be no history at all, the growth being discovered by accident. The cranial neoplasms, however, always have a chronic history of a palpable, and often visible lump.

Usually the mass is not tender to pressure, but it may become so. It is hard to the touch in most cases, and the scalp tissues move more freely over it, if the lesion is in the vault. In a few cases, however (Zajackowski, 1901; Schöne, 1905), the center of the tumor is soft and pulsating, while the periphery consists of a ring of hard bone raised above the level of the neighboring skull. These characteristics occur in the extracranial types of hemangioma also (e.g. Mauguiere, 1920; Bucy and Capp, 1930) and constitute one of the forms of the "aneurysm of bone" of earlier authors. There is no dilatation of the vessels in the soft tissues over the lesion, and no audible bruit. There is no alteration of hair growth in the scalp overlying these neoplasms when they occur in the vault.

At first there is merely the story of a lump. Later on, the patient usually complains of headaches, which gradually become more severe and frequent as the mass expands. In female patients the severity of the headaches may be paroxysmal and related to the onset of menstruation. The headaches may be the result of pressure on the dura (q.v.). If the tumor becomes sufficiently large, other symptoms may appear as the result of pressure on the underlying brain. However, with those hemangiomata located in the vault, neurological symptoms and signs are most unusual, as the tendency is more towards external than internal expansion. The intracranial pressure is rarely elevated, and the chemistry of the cerebrospinal fluid remains within normal limits.

In the case of the orbital and petrotemporal lesions, however, local symptoms and signs appear much earlier in the history. In the case of the former there may be proptosis, diplopia and other visual disturbances, while the latter often produce deafness, tinnitus and facial paresis of lower motor neurone type. The petrous hemangioma may encroach on the external auditory meatus, and produce a mass visible at otoscopic examination (e.g. McKenzie, 1939); in other cases it may involve the labyrinth, and result in giddiness, ataxia, nystagmus and other signs of vestibular disturbance. Such signs are unusual, however, as the labyrinth is well protected by the denseness of its ossific capsule. The appearance of unilateral middle ear deafness and homolateral lower motor neurone facial paresis, in the absence of other signs except perhaps slight vestibular disturbances, should suggest a petrous hemangioma as one of the possibilities, especially if the history is a long, but slowly progressive one.

4. ROENTGENOGRAPHIC APPEARANCES

The first roentgenographic description of an osseous hemangioma was published by Hitzrot (1917), but no correct preoperative diagnosis of cranial hemangioma was made until Kaplan and Kanzer (1939) reported a case. Since then the correct diagnosis has been made from the roentgenographic appearances on a number of occasions (e.g. Abbott, 1941; Rowbotham, 1942; Paul, 1946) including the author's case. The roentgenographic features of primary hemangioma of the skull will be considered in some detail, as they constitute the essential means of diagnosis.

FIG. 1. Posteroanterior roentgenogram showing the cranial defect (indicated by arrows) in the left frontal region. Note the erosion of bone and the delicate stippling within the eroded area.

Hemangiomata of the vault may be satisfactorily demonstrated in the usual posteroanterior and lateral views, while those occurring in the occipital and petrous bones require the use of the Towne-Twining position. Occasionally, Stenvers' position or Schüller's position may also be of use.

The characteristic features are at once apparent on inspection of the films. There is a rounded area of rarefaction, usually in the parietal or frontal region, which has a peculiar (and diagnostic) "honeycomb" appearance (Fig. 1 and 2). The shape of this area is round or oval, never serpiginous, although the borders of the rarefied area are often irregular. There is no "punched out" appearance, and rarely is there much in the way of osteosclerotic reaction in the surrounding bone, except perhaps in the pulsating types of tumor. The interstices between the bony trabeculae vary in size, and some may be quite large; these contain

FIG. 2. Lateral view of the skull of the same patient showing similar appearances. Note the absence of reactive sclerosis, and the absence of vascular changes in the bone.

"blood cysts" (q.v.). The bony fenestrations within the rarefied area are usually not very dense. There are no dilated vascular channels or diploic lakes in relation to the tumor, although occasionally its derivation from a diploic vein may be apparent in the films. The above features are well demonstrated in a roentgenogram of the piece of bone removed by block resection from the author's case (Fig. 3). A diploic vein can be seen entering the tumor, which appears to arise from it like a small shrub branching out from its stem.

If the lesion is visualized in tangential views (Fig. 4) another characteristic feature is demonstrated. This is the so-called "sunray" appearance common to all hemangiomata of bone, which is produced by the radiating bony trabeculae. It will be noted that these trabeculae are arranged in a radial manner, tending to arise from a com-

mon center; this is in contrast to the appearance of calvarial meningiomata in which the striations tend to be parallel with one another (Sosman and Putnam, 1925; Sosman, 1937). The radial disposition of the trabeculae may sometimes be apparent in direct views of the tumor, although more often there is merely an irregularly fenestrated meshwork, as in Figure 3.

The tangential views also reveal the expansion of the calvarium which occurs. Erosion of the outer table alone is the commonest finding, with external expansion of the tumor, and preservation of the integrity of the inner table. This can be seen in the profile view of the piece of bone removed from the author's case (Fig. 5). As the

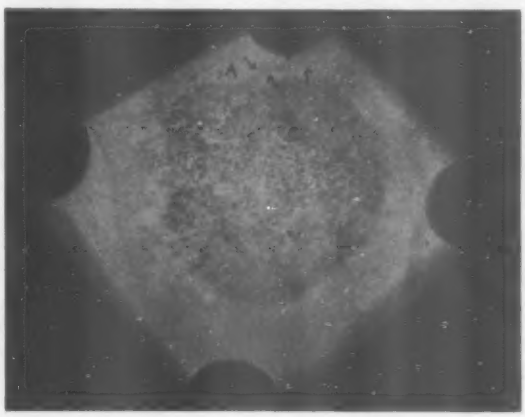

FIG. 3. Roentgenogram of piece of bone removed in the author's case. The honeycomb appearance is well demonstrated, and the parent diploic vein can be seen entering the tumor at A.

lesion grows, ballooning of the internal table occurs, and it may become eroded. However, intracranial expansion and destruction of the internal table always lags far behind the changes in the external table, and therefore the signs and symptoms of a space occupying lesion are extremely unusual with cranial hemangiomata.

Another feature apparent from profile views of the tumor is the absence of reactive sclerosis or hyperostosis in relation to it. Here again the appearance differs from that of meningiomata, which commonly produce marked hyperostotic changes in

FIG. 4. Tangential roentgenogram of the same patient's skull, showing the radial striation so characteristic of these tumors.

the related portion of the cranium.

Air studies are of little importance in these tumors. A normal ventricular system is demonstrated by pneumoencephalography or pneumoventriculography, and no displacement of the intracranial contents occurs. Occasionally, if the hemangioma is large, the introduction of air over the cortex may reveal a small dimple in the brain surface, where it has been indented by the internal expansion of the tumor. These special procedures are quite unnecessary as the appearance in the plain roentgenograms is characteristic and sufficiently diagnostic on careful inspection.

To sum up, the characteristic roentgenographic features of primary calvarial hemangioma may be enumerated as follows:

IN DIRECT (EN FACE) VIEWS

1. A circumscribed, oval, rarefied area, usually in the parietal or frontal regions.

FIG. 5. Profile roentgenogram of the bone removed from the author's case. The radial striation and the proportionately greater erosion of the outer table are well seen.

2. A "honeycomb" appearance within the rarefied area.
3. Absence of reactive sclerosis or vascular changes in the skull.

IN PROFILE VIEWS

1. Radial striation, producing a "sunray" appearance.
2. Erosion of the external table, with expansion more marked externally than internally.
3. Preservation of the integrity of the inner table of the skull.
4. Absence of reactive hyperostosis.

Hemangiomata in regions of the skull other than the vault are not so characteristic in their roentgenographic appearances. If the lesion is well forward in the frontal bone, it may encroach upon the frontal sinus, producing deformity of its posterior wall, as in Rowbotham's (1942) Case 3. In other cases the roof of the orbit may be depressed, while in those hemangiomata arising from the orbital plate of the frontal bone expansion occurs mainly at the expense of the intraorbital contents, resulting in proptosis and venous congestion within the eyeball, and the development of unilateral papilledema. Such lesions, in some cases, may be difficult to differentiate from the sphenoidal meningiomata of the type described by Cushing and Eisenhardt (1929) and Cushing (1929). However, in the case of the hemangiomata, the reactive changes in the roof of the orbit are not so sclerotic, as is the case with the meningiomata.

In the case of hemangiomata of the petrous temporal bone, erosion of the petrous may be seen in films taken in the Towne-Twining position. Usually the labyrinth is preserved, on account of its sheath of very dense bone. Trabeculation in this site is seldom apparent, but again, there is little or no reactive sclerosis in the neighboring bone. Lateral views of the mastoid region, or films taken with Stenvers' or Schüller's projections, may sometimes reveal the lesion if it has encroached upon the external auditory meatus or is invading the mastoid process.

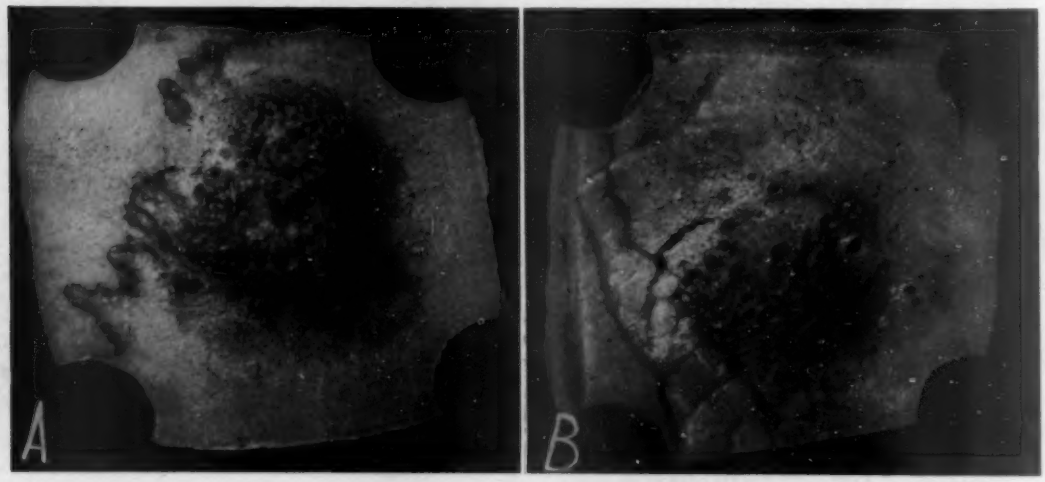

FIG. 6. *A*, external view of the bone removed from the case described. The coronal suture is visible on the left, and to the right of this is the neoplasm. Note the hollows which are occupied by blood-filled cysts. *B*, intracranial aspect of the same tumor. Note the lesser expansion of the tumor on this aspect, and the larger size of the cystic spaces. There is obviously no attachment to the dura.

5. PATHOLOGY

Macroscopically, as seen at operation, primary hemangioma of the calvarium appears as a hard, blue-domed lump on the skull, lying beneath the pericranium, which can be lifted off it. Against the bluish background of the tumor, which forms a distinct contrast to the white calvarium, deep red

blood cysts are apparent. These are distended while the tumor is in situ, but as soon an the bone is removed they collapse, producing crater-like depressions. If one of these cysts is pricked, dark venous blood oozes out. The cysts appear to be more conspicuous on the internal than on the external surface of the lesion. The macroscopic features are seen in Figures 6 and 7 and in Figure 8, which shows the cross-sectional aspects of the tumor, constituted by blood-filled spaces of varying size and depth. These are enclosed by the bony trabeculae which form such a prominent roentgenographic feature.

Histopathologically considered, hemangiomata of bone may be either of capillary or cavernous type. The capillary type is rare, especially in the skull. Nearly all the

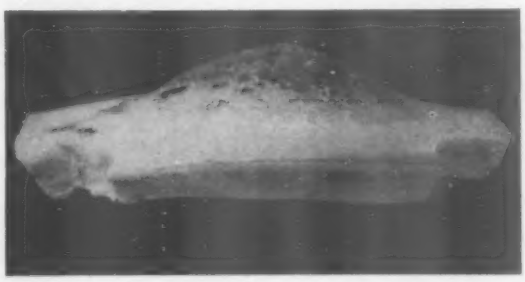

Fig. 7. Profile view of the same tumor.

hemangiomata of the skull that have been examined microscopically have proved to be of the cavernous variety. Tumors of this type (Fig. 9) consist of large blood-filled lacunae embedded in a connective tissue matrix: they are lined by large, flattened endothelial cells. They arise from one or more diploic veins, and their walls therefore do not contain the muscular or elastic tissue components characteristic of normal blood vessels. The bony trabeculae are not of neoplastic origin, as they are continuous with the osseous tissue of the adjacent skull, and their lamellae have an orderly pattern (Rowbotham, 1942). The osseous material lies outside the fibrocellular matrix which encloses the endothelial-lined spaces, whose pattern is the resultant of simultaneous processes of bone destruction and bone formation. Both osteoblasts and osteoclasts are numerous in relation to the bony spicules, and the remodeling they produce is a manifestation of the reaction of the bone to the presence of the neoplasm. The amount and cellular content of the stroma is the most variable feature of these tumors. In some cases many cells may be seen, and occasionally "nests" of lymphocytes are embedded in the stroma. Occasionally larger cells with vacuolated cytoplasm may be visible; these are the so-called "foam cells" and are undergoing fatty degeneration. Degenerative and necrotic changes may also be apparent in the osseous matrix

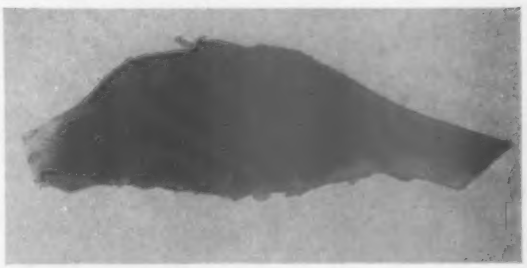

Fig. 8. Cross-sectional aspect of the tumor from the author's case. Here both outer and inner tables have become eroded. The absence of reactive sclerosis or hyperostosis is apparent. The whole tumor consists of blood-filled spaces.

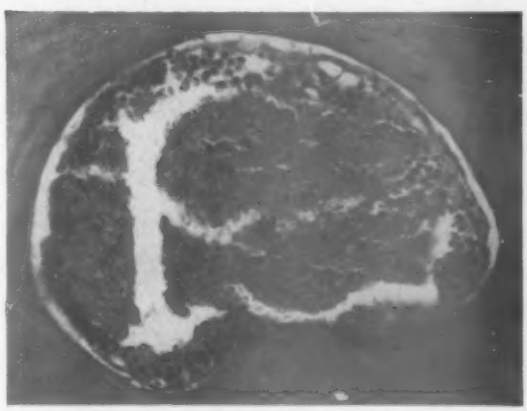

Fig. 9. Photomicrograph of primary cavernous hemangioma of the skull. The tumor consists of blood-filled spaces, separated by spicules of normal bone. The section shows one of these spaces, in which proliferation of the endothelial lining is apparent. (Hematoxylin and eosin, ×100.)

of the older and larger tumors, which have outstripped their blood supply.

Primary hemangiomata of the skull are always benign. No metastases have ever been reported, and there are no changes in regional lymph glands. The benign nature of these tumors is evident from the long histories of some of the cases that have been reported, notably those of Anspach (1937) and Rowbotham (1942). Histopathologic changes suggestive of malignancy have never been noted. The primary hemangiomata of the skull do not spread to involve the underlying dura or brain, and lateral expansion within the calvarium occurs to a far less extent than that at right angles to the bone, which is ballooned outwards in the manner previously described.

6. DIFFERENTIAL DIAGNOSIS

Clinically, the differential diagnosis is that of a hard lump on the head. The important distinctions that have to be made are those based on an examination of the plain roentgenograms of the skull.

Frequently, a primary hemangioma of the skull is diagnosed as a meningioma, as occurred in the case reported here. This is the result of cursory examination of the roentgenograms, as the "honeycomb" appearance is not seen in the meningiomata, although at times these may produce stippled, eroded areas. Moreover, although vertical striations occur in the meningiomata, they tend to be parallel with one another, not radially disposed as in the hemangiomata. Again, absence of reactive sclerosis or hyperostosis is another distinguishing characteristic, especially important in differentiating the orbitosphenoidal tumors. Also, in the case of the meningiomata, erosion of the skull occurs from within out, so that the internal table is affected more than the external table. The presence of enlarged vascular channels and diploic lakes in relation to meningiomata is another differential feature.

The presence of a rarefied area in the vault may be suggestive of the rare cranial cholesteatoma, or skull epidermoid. In these tumors, however, there is usually a palpable skull defect surrounded by a bony ridge, and in the roentgenograms the outline of the defect is usually serpiginous, and heavily sclerotic. If profile views are obtained, it will be seen that erosion of the internal table is more marked than that in the external table, in contrast to the hemangiomata. There is no striation and usually no honeycomb appearance, except in some cases where the outer table of the skull is partially eroded. The lesion is generally in the temporal region of the skull, and is much less frequently seen involving the bones of the vault. It arises within the diploe, but balloons out the inner table of the skull earlier and to a much greater extent than the outer table.

Specific or nonspecific osteomyelitis may produce rarefied areas in the skull, but the appearance is that of a diffuse mottling. Usually the areas are multiple, and serpiginous in outline. There is no lump on the skull, except in the case of Parrot's node, which occurs in some syphilitic infections of the skull. This is distinguished by the absence of striation, and the uniform density of its shadow in the roentgenograms.

Xanthomatosis (Schüller-Christian's disease) involving the skull produces rarefied areas in the vault, and occasionally lumps on the skull. However, these patches are commonly multiple and are clear, without any "honeycomb" appearance. The edges of the defects are clear and sharply defined, and the lesion could hardly be mistaken for primary hemangioma of the skull.

Osteoporosis circumscripta could be confused roentgenographically with hemangioma of the skull. There are circumscribed areas of rarefaction in the skull, which may be single, and the disease involves the outer table first. However, there is no "honeycomb" appearance and no radial striation. No lump appears on the skull. The same applies to the fibrosing type of osteitis.

Certain anemias may be accompanied by relatively localized areas of thickening in the skull, and the development of stria-

tions in the bone, when seen in profile views. In addition, in Cooley's anemia, there is an increase in the diploe, with thinning of the outer table. However, these conditions are seen only in young people, tend to be diffuse and bilateral in the skull, and there is no lump visible or palpable. Direct views do not demonstrate any circumscribed areas of erosion, and a blood count will reveal the characteristic anemia.

Schwartz (1939) suggests that certain types of osteitis fibrosa cystica may produce a picture similar to that of hemangioma. However, the general changes in the skull, and the absence of a mass and of "honeycomb" striations, readily distinguish the lesion.

Osteomata may produce isolated lumps on the skull, but the roentgen appearance is quite different. There is a more or less uniform sclerosis of the lesion in profile view, and no striation, while direct views demonstrate an area of increased density in the skull.

The osteolytic type of osteogenic sarcoma is unusual in the skull, but may give rise to a localized, pulsating swelling. Roentgenographically, however, there is involvement of inner and outer tables to an equal extent, and marked internal, as well as external, expansion of the skull. Striation is not well marked and is irregular, and there is no "honeycomb" appearance in the bone defect.

A localized area of erosion in the skull may be produced by expansion of an intracranial hemangioma or a cirsoid aneurysm. However, stereoscopic views reveal that the erosion is occurring from within out. There is no diploic expansion, no striation, and no "honeycomb" effect.

7. TREATMENT AND PROGNOSIS

Primary hemangioma of the skull has been treated both surgically and by radiotherapy.

Removal of the tumor was first attempted by Ehrmann (1847) on the second case reported. The lesion was removed, but the patient died of postoperative meningitis. The first case of successful removal with recovery of the patient was reported by Pilcher (1894), who excised the tumor piecemeal. Cushing (1923) performed a block resection of a primary hemangioma of the skull, and this has been successfully repeated on a number of occasions since, including the present case. Block resection removes the tumor intact, and obviates the serious risk of hemorrhage which is present when a direct attack is made upon the lesion. The cranial defect may be easily restored with a bone graft from the tibia, or better, from the internal table of the ilium.

In view of the relative ease and success of the surgical method, irradiation is seldom necessary, except in the case of the petrous hemangiomata. Here deep roentgen therapy (McKenzie, 1939) or radium (Irgens, 1939) may be employed. Some of the deep-seated orbital hemangiomata are also suitable for radiation therapy, although special surgical techniques (e.g. Paul, 1946) may facilitate their removal.

The prognosis with surgical removal by block resection is uniformly good. The outlook with radiation therapy is slightly less favorable. Without treatment the tumor proves fatal after a long period, as it encroaches on the intracranial contents, or may become necrotic, and ulcerate the skin. With the petrous and orbital hemangiomata the prognosis is always more grave, in view of the local loss of function that occurs. Tumors in these sites are more rapidly fatal than those in the vault, although, again, the history is usually relatively long.

CASE REPORT

R. H. (M6970), female, a Bible student, aged twenty-eight, was admitted to the Royal Prince Alfred Hospital on February 18, 1946, complaining of a lump on her head, which had been present for two years. For six months she had had headaches, which had become worse recently. She had difficulty in concentrating on her studies, but no other symptoms except some menstrual irregularity. The lump had been slowly increasing in size. While at school she had been struck on the head with a hockey

stick in the region concerned. Her previous health had been good.

On examination a hard, immobile lump was apparent in the left frontoparietal region. Measuring 2.5 by 3.5 by 1.5 cm., it was not tender, and did not pulsate. The scalp was movable over it, and there were no dilated vessels apparent over the lump. Complete general and neurological examination revealed no abnormalities. Blood count was normal. Lumbar puncture revealed normal cerebrospinal fluid at a pressure of 100 mm. of water.

Roentgenographic Examination. Posteroanterior and lateral plain roentgenograms of the skull were taken, and the following report was tendered: "There is a circular defect in the left frontal bone just anterior to the coronal suture. This has the typical stippled appearance of meningioma." However, careful inspection of the films revealed that the appearance of the lesion was not typical of meningioma, and tangential rays demonstrated an intradiploic lesion with radial striations. Pneumoencephalography revealed no intracranial disturbance as a result of the lesion, which was growing externally rather than internally.

Operation. With intratracheal ether as anesthetic, a left frontal flap was formed in the scalp, and the bluish dome of the tumor exposed. It was found to be subpericranial, and the pericranium was easily separated from the tumor. Four burr holes were introduced in the bone at the angles of a 5 cm. square enclosing the tumor, and united with Gigli saw cuts. The block of bone so delineated was easily lifted out, there being no attachment to the dura. There was a small dimple in the dura and brain about 0.5 cm. deep beneath the tumor, but this soon filled up after its removal. A flap of bone the size of the cranial defect was then prepared from the internal surface of the right ilium, and fixed in place in the skull, with catgut sutures. The scalp tissues were approximated in two layers in the usual way.

Following the operation the patient had an uneventful recovery, and was discharged on the eighteenth postoperative day with both the scalp and iliac wounds healing well, and the bone graft firmly in place. Since then she has been well, and free from headaches.

Pathologically the tumor was found to be a primary cavernous hemangioma of the left frontal bone.

Thanks are due to Mr. R. A. Money, Honorary Neurosurgeon at the Royal Prince Alfred Hospital, Sydney, for permission to report the case; and to Mr. S. Woodward-Smith, of the Department of Medical Artistry, University of Sydney, who is responsible for the photographic reproductions.

9. SUMMARY AND CONCLUSIONS

1. The general incidence of hemangioma of bone is 0.7 per cent of osseous neoplasms.

2. The general incidence of primary hemangioma of the skull is 0.2 per cent of osseous neoplasms, and 10 per cent of primary benign neoplasms of the skull.

3. Primary hemangioma of the skull is most frequent in the fourth decade, few cases being recorded before the thirtieth year.

4. Primary hemangioma of the skull is three times as common in females as in males.

5. The parietal bones are most frequently involved, and then the frontal bones.

6. The diagnosis of these tumors depends upon recognition of the characteristic roentgenological features, which are discussed.

7. The pathology of primary hemangioma of the skull is described. Nearly all cases are cavernous hemangiomata. The tumor is benign always.

8. The differential diagnosis is discussed.

9. Treatment may be surgical or by radiation therapy. Block resection, and replacement of the resulting defect by a tibial or iliac bone graft, is the preferable method of treatment in calvarial tumors. Irradiation is best employed for the petrous hemangiomata.

10. A case of primary hemangioma of the frontal bone is reported, with the roentgenographic findings, and treatment by block resection and replacement by iliac graft.

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CONVOLUTIONAL MARKINGS IN THE SKULL ROENTGENOGRAMS OF PATIENTS WITH HEADACHE*

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AREAS of diminished thickness of the calvarium appearing like digitations on the roentgenogram of the skull are frequently observed, and are commonly referred to as *convolutional markings*. They are believed to be due to the impressions of the cerebral convolutions on the inner table of the skull and are usually seen in the first two decades of life during the period of greatest growth of the brain. They first appear toward the end of the first year of life (Davidoff,[1] 1936) when the fontanelles and sutures begin to close, and then increase in prominence up to about seven years of age. They then are quite evident until about fourteen years of age, become less marked for a few years and increase somewhat during later adolescence. They are generally believed to be uncommon during adult life except when seen, along with atrophy of the sella turcica, as an indication of increased intracranial pressure.

In reading the roentgenograms of the skull taken routinely upon patients admitted to the Headache Clinic at Montefiore Hospital, convolutional markings were encountered with seemingly unusual frequency. In adult patients suffering from recently developed headaches the possibility that the presence of such markings might be reflecting increased intracranial pressure from brain tumor was of some concern. In addition the etiological significance that a high incidence of convolutional markings might have for the headache problem in general further warranted investigation. Information available in the literature regarding the incidence of digital markings in the normal adult individual or in patients suffering from headache is meager. Accordingly the present study was undertaken.

One hundred consecutive roentgen examinations of the skull made on patients from the Headache Clinic were sorted as to age and sex. A control group of skull roentgenograms from another 100 completely healthy subjects who did not suffer from headache were then made to match the age (by five year groups) and sex distribution of the headache patients. All the films were then studied for the presence of convolutional markings. When present these were graded from 1 to 4, 1 indicating minimal digital impressions on the inner table of the skull regardless of locality and 4 indicating maximal markings (Fig. 1). Upon subsequent investigation and follow-up examinations a diagnosis of organic neurological disease compatible with increased intracranial pressure was not made on any of the headache patients who had convolutional markings.

RESULTS

Two points of considerable value were immediately evident upon reviewing our figures, namely: (1) convolutional markings are quite commonly present in adults (45 to 46 per cent); and (2) they occur just as frequently in healthy adults without headaches as they do in adults who are healthy except for headaches.

Table 1 indicates the distribution of convolutional markings in each group as a whole. In view of the approximate equality of the total number of patients in each group who showed markings, the difference between the number of patients showing only 1 and those showing 2 degrees of

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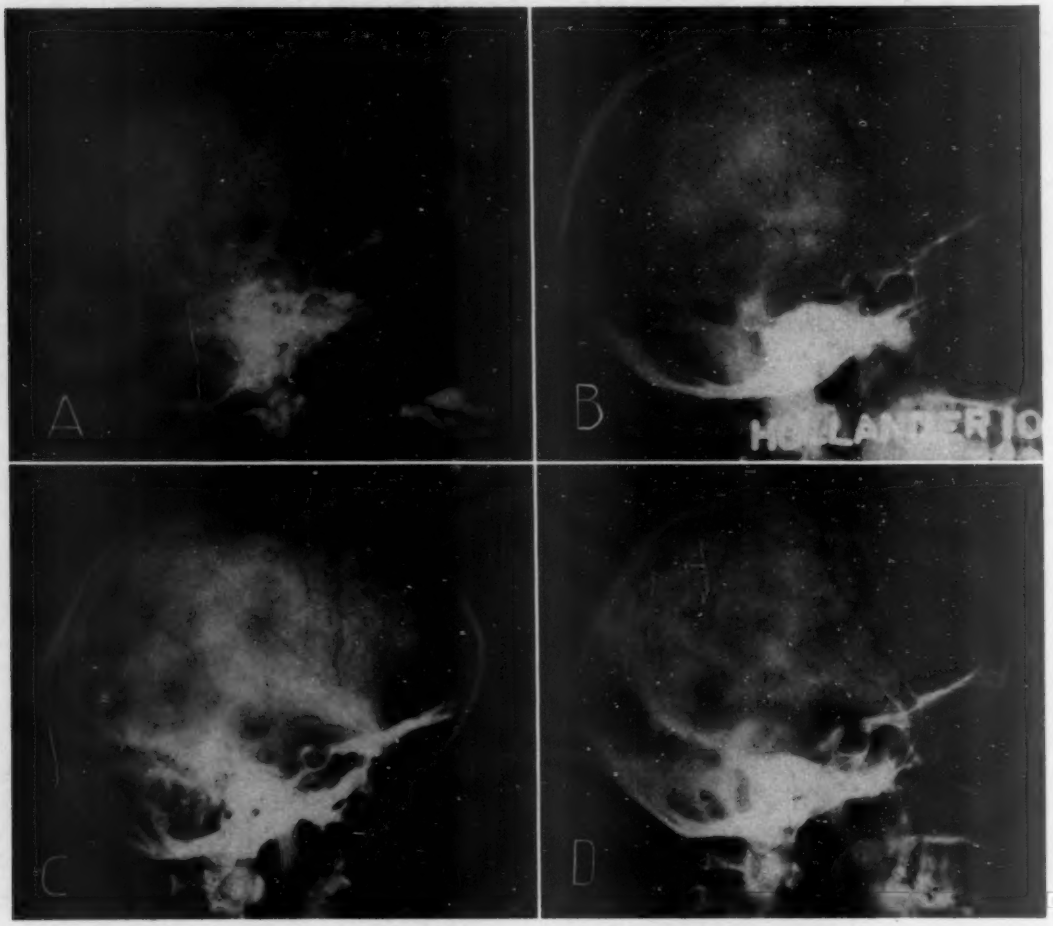

Fig. 1. Convolutional markings in adults graded from 1 to 4. (*A*) grade 1, female, aged thirty-five; (*B*) grade 2, male, aged thirty-nine; (*C*) grade 3, female, aged twenty-three; (*C*) grade 4, female, aged twenty-nine.

markings in the two groups is probably of no clinical significance. Thus the markings in roughly a third of all subjects in each group were only slight in degree (grade 1 or 2). About 10 per cent of each group showed moderate to marked digital impressions (grade 3 or 4).

Table I

INCIDENCE OF CONVOLUTIONAL MARKINGS IN ENTIRE SERIES

Degree of Convolutional Markings	1	2	3	4	Total
Headache patients	17	17	10	2	46
Normal controls	27	9	8	1	45
Total	44	26	18	3	91

In Table II convolutional markings appearing in each group are classified by age and sex. The age and sex distribution of the 100 patients originally used does not show an even spread in each five or even ten year age group. The incidence of convolutional markings is therefore represented in each age group and in each sex by the percentage of the total number of patients of that sex in a given age group who showed markings. A ten year age grouping was used arbitrarily for simplicity. This distribution is also demonstrated graphically in

TABLE II

DISTRIBUTION IN EACH GROUP BY AGE AND SEX

Distribution of the 100 Patients in Each Group				Headache Patients with Convolutional Markings						Normal Controls with Convolutional Markings					
Age yr.	Male	Female	Total	Male	Per cent of males in 10 year group	Female	Per cent of females in 10 year group	Total	Per cent of 10 year group	Male	Per cent of males in 10 year group	Female	Per cent of females in 10 year group	Total	Per cent of 10 year group
21–25	9	5	14	3	43	4	50	7	46	3	29	4	83	7	54
26–30	5	7	12	3		2		5		1		6		7	
31–35	3	13	16	1	44	11	70	12	63	0	33	9	65	9	56
36–40	6	10	16	3		5		8		3		6		9	
41–45	0	10	10	0	67	6	53	6	56	0	0	7	50	7	50
46–50	3	5	8	2		2		4		0		2		2	
51–55	2	11	13	0	0	4	27	4	19	1	33	2	10	3	19
56–60	4	4	8	0		0		0		1		0		1	
over 60	0	3	3	0	—	0	0	0	0	0	—	0	0	0	0
Total	32	68	100	12	38	34	50	46	46	9	28	36	53	45	45

Figure 2. Although there are some differences between the normal and headache groups when graphed according to sex, the age distribution of patients with convolutional markings when sex is disregarded is very similar. Markings are most frequent in each of these adult groups in the third, fourth and fifth decades of life, being present in about half of all such patients. In the sixth decade the incidence falls off rapidly, and no markings were noted in 3 female patients in each group over sixty years of age. The difference between the curves for males may be more apparent than real inasmuch as the high percentage of markings in headache males in the forty-one to fifty year group compared to the absence of markings in this age group in the controls is based in each instance on the examination of only 3 subjects in that particular age group. The chief difference between the curves for females is the higher incidence of markings in the control twenty-one to thirty year age group.

When the subjects in both the headache and the control groups who show convolutional markings are classified together as a unit by sex and age, as in Table III, certain consistent differences in sex incidence are apparent. These are graphically demonstrated in Figure 3 where it is noted that

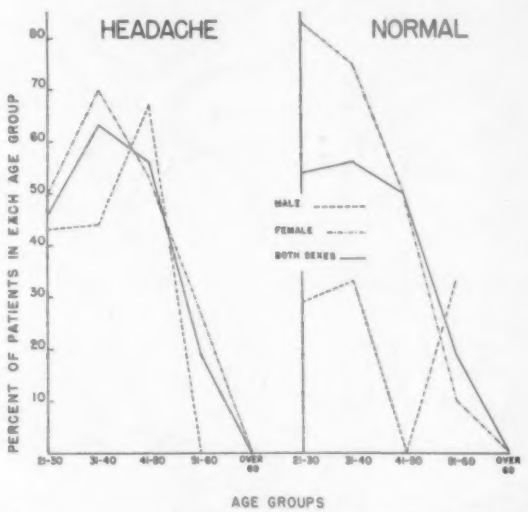

FIG. 2. Age and sex distribution of convolutional markings in each group.

TABLE III

AGE AND SEX DISTRIBUTION IN COMBINED HEADACHE AND CONTROL GROUPS

Age yr.	Male	Per Cent of Males in 10 Year Group	Female	Per Cent of Females in 10 Year Group	Total	Per Cent of Patients in 10 Year Group
21–30	10	36	16	67	26	50
31–40	7	39	31	67	38	59
41–50	2	33	17	57	19	53
51–60	2	17	6	20	8	19
over 60	0	—	0	0	0	0
Total	21	33	70	51	91	46

in the third, fourth and fifth decades of life the incidence of markings in females is almost doubled that in males of comparable age groups. In the sixth decade of life the female and male incidence is approximated and at a much lower level.

In Table IV convolutional markings are

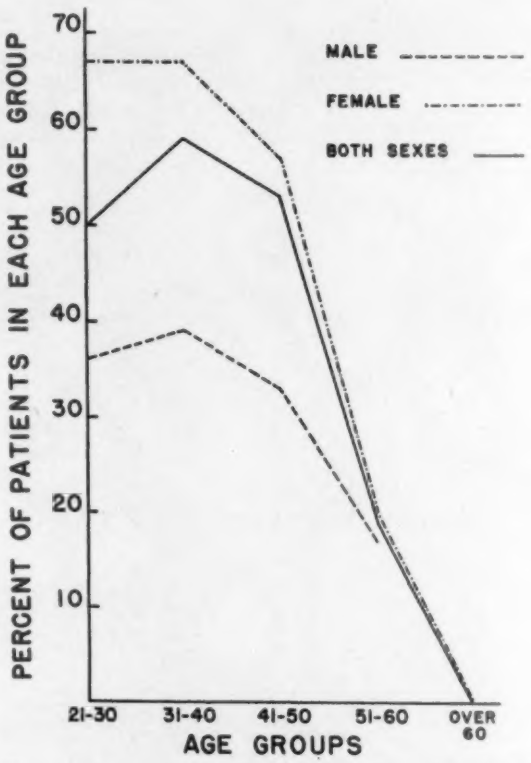

FIG. 3. Age and sex distribution of convolutional markings in both groups combined.

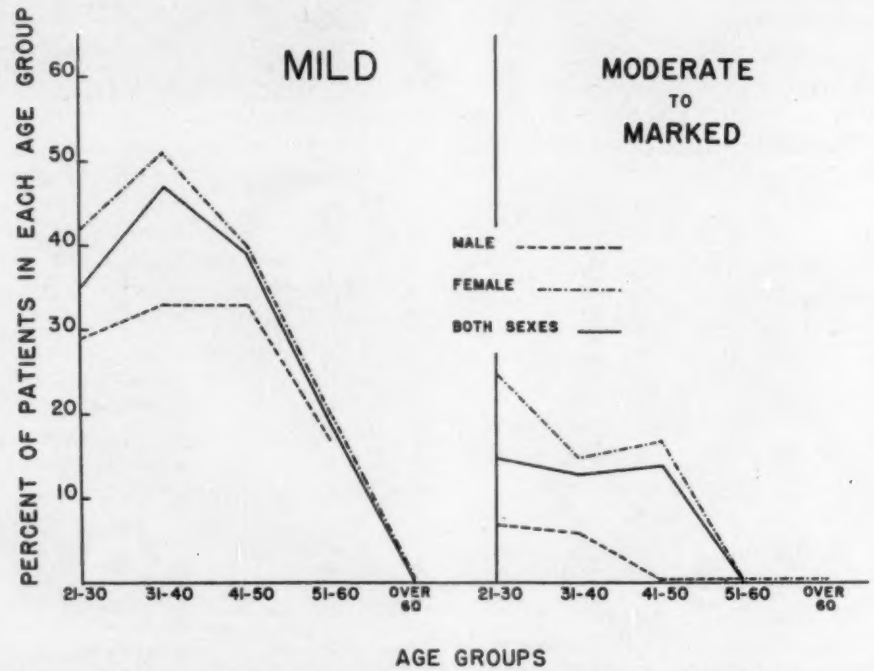

FIG. 4. Age and sex distribution of mild (grades 1 and 2) and moderate to marked (grades 3 and 4) convolutional markings in the combined headache and normal groups.

TABLE IV

AGE SEX AND DEGREE DISTRIBUTION OF CONVOLUTIONAL MARKINGS

Degree of Convolutional Markings	1			2			3			4		
Headache Patients												
Age yr.	Male	Female	Total	Male	Female	Total	Male	Female	Total	Male	Female	Total
21–25	1	0	1	1	2	3	1	2	3	0	0	0
26–30	0	1	1	2	0	2	1	1	2	0	0	0
31–35	0	4	4	1	4	5	0	2	2	0	1	1
36–40	0	3	3	2	2	4	0	0	0	1	0	1
41–45	—	3	3	—	0	0	—	3	3	—	0	0
46–50	1	2	3	1	0	1	0	0	0	0	0	0
51–55	0	2	2	0	2	2	0	0	0	0	0	0
56–60	0	0	0	0	0	0	0	0	0	0	0	0
over 60	—	0	0	—	0	0	—	0	0	—	0	0
Total	2	15	17	7	10	17	2	8	10	1	1	2
Normal Controls												
21–25	2	2	4	1	0	1	0	2	2	0	0	0
26–30	1	4	5	0	1	1	0	0	0	0	1	1
31–35	0	7	7	0	0	0	0	2	2	0	0	0
36–40	2	3	5	1	1	2	0	2	2	0	0	0
41–45	—	4	4	—	2	2	—	1	1	—	0	0
46–50	0	0	0	0	1	1	0	1	1	0	0	0
51–55	0	1	1	1	1	2	0	0	0	0	0	0
56–60	1	0	1	0	0	0	0	0	0	0	0	0
over 60	—	0	0	—	0	0	—	0	0	—	0	0
Total	6	21	27	3	6	9	0	8	8	0	1	1

classified by degree according to age and sex in the headache and the control groups. In Figure 4 the incidence of slight convolutional markings (1 or 2 degrees) in both headache and control subjects is graphed by age and sex, and is compared with a

similar graph for patients of both groups having moderate to severe markings (3 or 4 degrees). It is apparent from this that the female sex predominance of markings exists regardless of the degree of convolutional markings. It is also evident that slight degrees of markings are likely to be present in about half of the subjects unclassified by sex in each of the third, fourth or fifth decades of life. The incidence of moderate or severe degrees of markings is much less but is also highest in the third, fourth and fifth decades of life than later on and occurs in about 15 per cent of all patients in these age groups.

COMMENT

Opinions expressed in the literature have varied concerning the significance of convolutional markings in the adult. Although many authors[2] have more recently recognized their occasional occurrence without pathological significance in the normal adult, not a few[3] view their presence with apprehension if not with pathognomonic indication of increased intracranial pressure. Statistical estimates of the incidence of digital impressions in adults not suffering from increased intracranial pressure have been found in only two reports in the available literature. Guardabassi and Salsano[2e] state that they may occur in 20 per cent of normal skulls. Perhaps the most extensive analysis of convolutional markings in the adult has been made by Ritter[2g] who examined for digital impressions the skull roentgenograms of 1,058 patients none of whom could have been suspected of having increased intracranial pressure. He stated that convolutional markings were scarcely ever encountered in patients over fifty years of age. He also noted that females had a greater incidence in comparable ages than males. There was no relationship noted by him between their incidence and the thickness of skulls. Between the ages of twenty and thirty-nine about 11 per cent of males and about 24 per cent of females in his group showed markings. Over the age of fifty they were present in about 3 per cent of males.

The results of our investigation indicate that they may be found without pathological significance with even greater frequency than mentioned by these authors, namely as high as in 45 per cent of all subjects, or in at least half of all females from the third through the fifth decades of life. In such rates of frequency, they are present in only slight degrees of intensity, but more severe manifestations may occur without significance in fully 15 per cent of all subjects in these age groups and especially in females.

There is no indication, moreover, that convolutional markings are more frequent than normal in patients suffering from headache in the absence of other evidence of increased intracranial pressure. Thus, the isolated appearance of convolutional markings in patients suffering from recently acquired headache without confirmatory positive clinical evidence of increased intracranial pressure need not necessarily be viewed with alarm, unless, of course, definite atrophy of the sella turcica accompanies it. In none of the headache or control patients could such atrophy be demonstrated.

CONCLUSIONS

1. Convolutional markings in skull roentgenograms are not seen more frequently in patients suffering from headache without other evidence of increased intracranial pressure than in healthy control subjects without headache.

2. Convolutional markings may occur in individuals in their third, fourth or fifth decades of life to a slight degree without pathological significance in half the skull roentgenograms taken in females and in a third of those taken in males. More severe markings may be present without significance in about 15 per cent of skull roentgenograms taken in all patients in these age groups.

3. Slight degrees of convolutional markings may occur even in the sixth decade of life in nearly 20 per cent of all skull roentgenograms without pathological significance.

4. Convolutional markings in the adult

occur more frequently in the female than in the male.

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A PRACTICAL SERIALOGRAPH FOR INTRACRANIAL ANGIOGRAPHY*

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INTRACRANIAL angiography is rapidly assuming a most important role in the diagnosis of intracranial disease. In past years the main drawback to the popularization of this procedure was in the use of thorotrast, a radioactive colloidal solution of thorium dioxide which is retained permanently in the reticulo-endothelial system of the patient and which also has been shown by Ekström and Lindgren[5] to produce cerebral injuries in many cases. The substitution by Gross[6] of diodrast for thorotrast as the opaque medium for visualization of the intracranial vasculature has removed that drawback to this valuable diagnostic procedure now widely used.

Intracranial angiography, because of the great wealth of information which it furnishes in many intracranial lesions, has now become an indispensable diagnostic procedure in the diagnosis of such lesions. At Montefiore Hospital this procedure has been carried out 148 times during the past fourteen months by combined teams from the departments of roentgenology and neurosurgery.‡

To facilitate and to meet the roentgen requirements of this procedure, I have designed a serialographic device which has been in frequent use at the hospital for the past fourteen months. This device has proved practical and simple to operate.

Since construction of the device is simple and since it can be built inexpensively by the carpenter shop of any hospital, it was thought worth while to describe the construction and operation of this device.

Cerebral arteriography was first described by Egas Moniz[2] in 1927. In 1934 he[4] published a monograph on cerebral angiography in which he described the normal distribution and appearance of the intracranial blood vessels, the diagnosis of cerebral tumors as depicted by the pathologic vascularization of tumors, the localization of cerebral tumors by displacement of cerebral vessels, the direct visualization of cerebral aneurysms and angiomas, as well as studies on brain abscesses and congenital hydrocephalus.

Since then a considerable European and American literature has grown up on the subject of intracranial arteriography and angiography which repeatedly stresses the indispensability of the procedure in the diagnosis of intracranial lesions.

The importance of intracranial angiography has become widely accepted and it is now being introduced in many institutions.

Intracranial angiography presents, however, a number of technical difficulties which must be mastered in order to derive the greatest amount of clinical information from the procedure.

TECHNIQUE OF INTRACRANIAL ANGIOGRAPHY

Introduction of the opaque medium into the intracranial circulation may be accomplished under direct vision after surgical exposure of the carotid, vertebral or subclavian arteries, or by surgical exposure of the radial artery and introduction of a urethral catheter through the radial artery into the vertebral artery (Radner[3]) or by percutaneous injection of the opaque medium into the vertebral or common carotid arteries.

Both methods of preparation of the

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patient for the injection of the opaque medium, as well as roentgenography of the intracranial vascular system during the very few seconds that the vessels remain opacified present several problems:

1. In those cases in which the percutaneous injection method fails for one reason or another, the patient must be taken to the operating room, where the incision and introduction of the needle can be carried out by the neurosurgeon with the necessary sterile precautions.

2. The patient must then be moved back to the roentgen department and roentgenographed without removal from the stretcher and without contamination of the operative field.

3. Lateral and anteroposterior angiograms must then be obtained while the patient remains in the supine position with the head in the normal position and without the necessity of turning the patient's head to either side, for the lateral roentgenograms. Efforts to obtain a true lateral view of the skull with the patient lying in the supine position on the conventional roentgen table, propped up with pillows under one shoulder, are usually not successful and are most uncomfortable for the patient.

There is great danger of contaminating the operative field during the effort to rotate the patient for the roentgenographic examination in this position.

4. There is also the great likelihood of dislodging the needle from the artery during this maneuver, particularly when the percutaneous method is employed.

5. Stereoscopic anteroposterior and lateral views are most desirable and should be obtained, if possible, since stereoscopic views are invaluable in the study of the abnormalities of the intracranial vasculature, produced by many of the intracranial lesions.

6. The examination of the intracranial vasculature should not be limited to the arteriographic phase but should include phlebograms of the superficial and deep intracranial veins, or so-called phlebograms of the first and second phase.

7. The neurosurgeon and the roentgen technician must be protected from the direct roentgen-ray beam and scattered roentgen radiation.

8. The device must be solidly constructed and not subject to undue vibration during the rapid removal and replacement of the exposed cassettes.

9. Construction of the device must be inexpensive and its operation fairly simple.

REVIEW OF THE LITERATURE ON DEVICES FOR ARTERIOGRAPHY AND ANGIOGRAPHY

Review of the literature on intracranial arteriography and angiography reveals the employment of a number of devices for this type of work.

Egas Moniz[8] (1933) made use of the Caldas carousel. This is essentially a rotating round table with a lead covering in which there is an opening for the successive introduction of each of six cassettes. In this way he was able to obtain six roentgenograms in six or seven reconds.

Later, as reported by Sanchez-Perez,[10] Egas Moniz used an "escomoteador," which is a box with an aluminum lid in which are placed three cassettes, containing roentgen films separated from each other by lead plates.

Rencz[9] (1936) constructed a stereoroentgenographic device in which he used two roentgen tubes operated simultaneously, with which he could obtain a stereoscopic pair of films during the arterial phase of the intracranial circulation. He called his device the stereo-arteriograph.

Dyes[3] (1941) devised an assembly for simultaneous roentgenography of the skull in the anteroposterior and lateral positions by using two roentgen tubes and two transformers.

Sanchez-Perez[10] (1943) designed a portable cranial seriograph, consisting of a box which holds three 10 by 12 inch cassettes, which are successively elevated into position beneath a Lysholm grid by vertical steel springs. The unexposed films are protected by interposed sheets of lead.

Holm[7] (1944), with the aid of a grant from the Rockefeller Foundation, devised a "cinematograph," with which he was able to expose eight to sixteen frames per second.

None of the above mentioned devices completely fulfills the practical features desirable in the practice of intracranial angiography.

Holm[7] writes with reference to his cinematograph that the technical difficulties in making the apparatus have been enormous and that not all of them have yet been solved. The greatest is the inability to secure sufficiently clear pictures. The reproductions are clear only within a central zone, 15 cm. in diameter. There are always details of interest outside this central zone which therefore are not reproduced with sufficient clarity. He states that he has recorded saccular aneurysms of the carotid artery, which on ordinary films were approximately pea-sized but which could not be definitely diagnosed on the cinematographic film due to lack of clarity.

The other above mentioned devices do not provide for or do not include one or more of the practical and desirable features previously discussed, namely, protection of the neurosurgeon and roentgen technician from roentgen radiation, difficulty in positioning the patient for roentgenography, with the attendant danger of contamination of the operative field, difficulty or inconvenience in obtaining both anteroposterior and lateral views of the skull, lack of provision for obtaining stereoscopic views and lack of provision for roentgenography during the arterial as well as during the venous phases of the intracranial vascular circulation.

A device for intracranial angiography which incorporates the above mentioned desirable features is not available at this time commercially. A combination serialograph-table-stretcher device was designed, therefore, by the writer, which has been functioning satisfactorily and which has been in frequent use for the past fourteen months at Montefiore Hospital.

The device can be built very inexpensively and its construction is as follows:

CONSTRUCTION OF SERIALOGRAPH

The apparatus consists of two parts: an auxiliary mobile table (Fig. 1); the serialographic assembly (Fig. 2).

The table is of very simple sturdy design, employing standard lumber in its construction. It is so mounted on the mobile iron pipe frame that it can be lifted and locked in a raised position when it is to be moved, or quickly lowered to the floor at rest. A sliding shelf (*3*) can be pulled out to extend the table top at its head. A deep foam-rubber mattress (*4*) rests on the table top; a smaller pillow (*5*) of the same material is used to support the patient's head when the head shelf is extended.

The table (Fig. 6) is elevated by foot pressure against the long wooden plank pedal lever (*6*) which by means of link rods (*8*) actuates a pair of auxiliary plank levers (*7*) which bear against the undersurface of the table top to lift it upward. The table is maintained in a raised position by the construction (Fig. 5) built into its underside. The table is lifted by levers (*6*) and (*7*) working in conjunction; while the foot is still bearing down on pedal lever (*6*), handle (*9*) is pushed in, causing the cleats at all four corners to move outward, coming to rest on top of the iron frame. The elevated table is thus supported firmly on the cleats which in turn rest upon the frame. To lower the table, pedal lever (*6*) is stepped upon, handle (*9*) pulled out to withdraw the cleats, allowing the table to be gently lowered, guided by foot pressure against the pedal. The patient can thus be transported, on this combination stretcher-table, from the operating room to the roentgen department.

The mobile serialograph itself is positioned against the table. The platform (*10*) on which the patient's head is to rest can be raised or lowered by crank (*11*) to accommodate anatomical variations, and to facilitate various roentgenographic techniques. The platform is cranked to the desired height, the extendable head shelf (*3*) on the table is slid back, pillow (*5*) removed, and the patient's head allowed to rest upon the platform. The table and serialograph are now locked together by long threaded rods (*12*) on both sides of the serialograph base, which fit into slotted plates (*13*) on the table legs. The wing-nuts on the threaded rods are

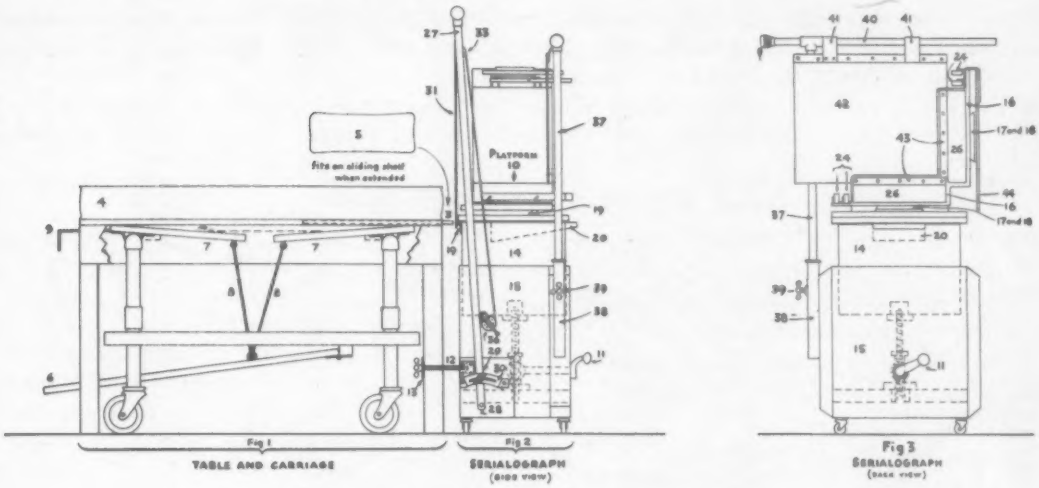

Fig. 1. Side view of table-stretcher combination.
Fig. 2. Side view of serialograph.
Fig. 3. Back view of serialograph.

now tightened, making the table and serialograph virtually a single rigid unit.

The Serialograph (Fig. 2, 3, 4). The base consists of two wooden boxes, one of which (*14*) nests within the other (*15*). This inner box can be raised or lowered within a range of approximately 12 inches by a screw-jack mounted within the outer box, operated by crank (*11*).

On top of this base assembly rests the serialograph proper, comprising two identical cassette tunnels (*16*) constructed of three-ply lead-lined rayboard (except for the platform

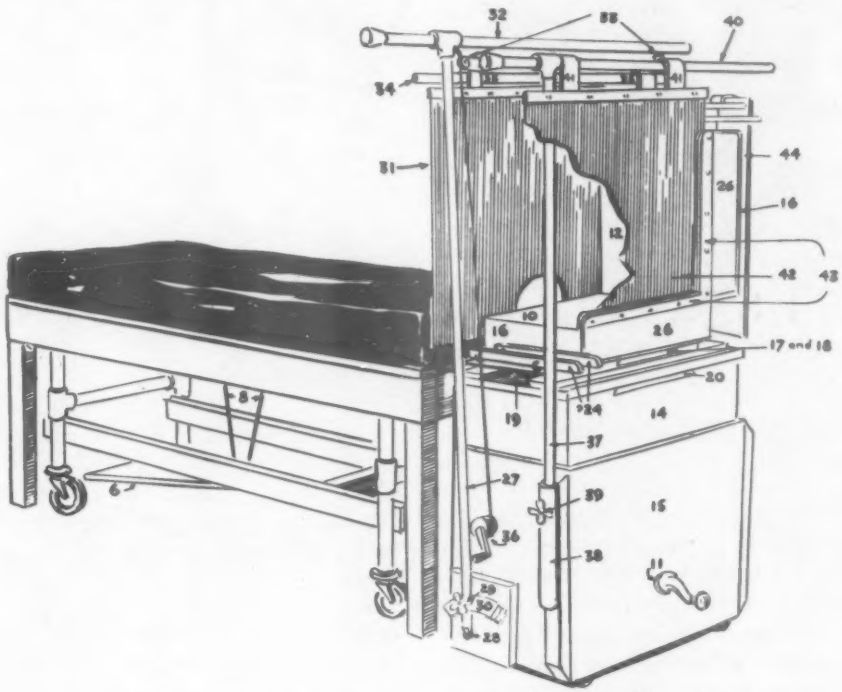

Fig. 4. Diagram of table-stretcher-serialograph combination.

No. 10 through which radiation must freely pass). These tunnels are mounted at right angles to each other. At the back of each of them there is mounted a bevel-edged board (*17*) which fits tongue-and-groove fashion into drawn out to tilt the serialograph forward (it is hinged at the front) providing desirable angulation for vertical beam anteroposterior head roentgenography (Fig. 9).

Cassette Tunnel. Figures 7 and 8 are detailed views of the cassette-changing mechanism of the serialographic assembly. The tunnel is deep enough inside to accommodate three standard 10 by 12 inch cassettes, stacked on top of each other. Cassettes No. 1 and 2 are fitted with 1/32 inch thick lead trays to stop roentgen radiation from reaching the cassette beneath. Each cassette is wrapped with a tape of 2 inch gauze, forming a handle for pulling the cassettes out of the tunnel during serialography. These tapes are either numbered in

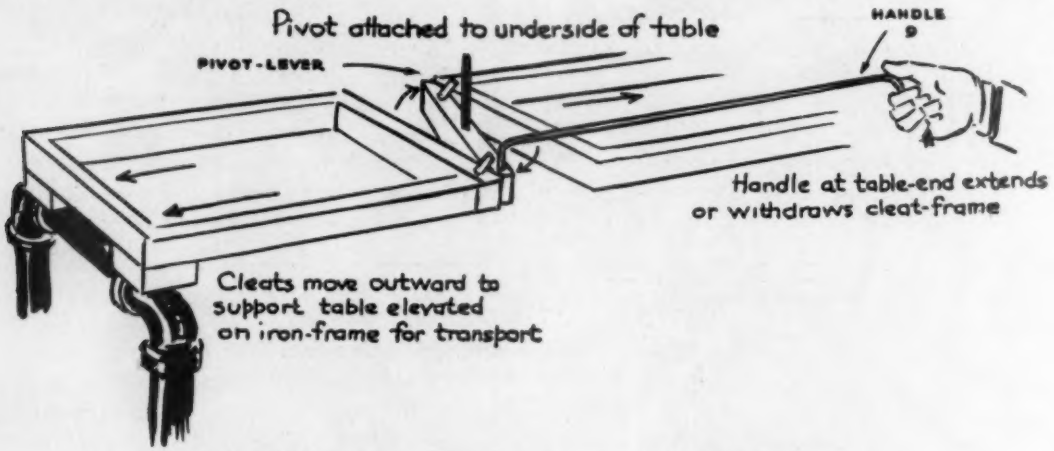
Pivot attached to underside of table
PIVOT-LEVER
HANDLE 9
Handle at table-end extends or withdraws cleat-frame
Cleats move outward to support table elevated on iron-frame for transport

Fig. 5. Diagram to show table elevating mechanism.

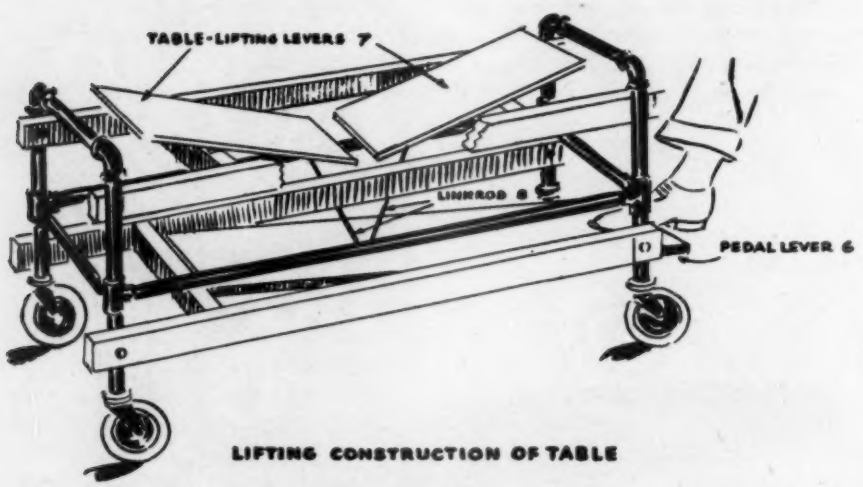

Fig. 6. Diagram to show table elevating mechanism.

a mating bevelled channel (*18*) in the baseboard. This sliding arrangement permits the serialograph to be moved toward or away from the table to accommodate patients of various statures. A second tongue-and-groove fitting (*19*) on a second board (directly beneath) lies at right angles to the first one, thus permitting a wide range of lateral and longitudinal shift. The combination of these two movements affords ample flexibility for positioning the serialograph. A wooden wedge (*20*) can be

sequence, or they may be color-coded for quick identification. At Montefiore Hospital the order is red, white and blue—an easily remembered sequence.

Sliding in angle channels (*21*) on either side which bring it directly against the underside of the top of the cassette tunnel is a Lysholm grid (*22*). These channels are designed to accommodate the grid in either its long (12 inch or short (10 inch) dimension. For lateral roentgenography done with a horizontal roentgen-ray beam the grid is inserted the long way. For anteroposterior roentgenography, however, the serialograph is angled to tilt the head forward, and since the platform, grid, and cassettes are in parallel relationship, the vertical lead strips of the grid (which run longitudinally in the 12 inch dimension in some of the grids) would also be angled, resulting in a partial cutoff of the roentgen-ray beam. In anteroposterior roentgenography, therefore, when using a grid in which the lead strips run longitudinally (in the 12 inch dimension) the grid is inserted in its shorter dimension so that no cutoff effect is encountered. The detail view of the channels in Figure 8*A* shows how the grid is held in either position. Since the channels are made of light aluminum, the filtration of that part of them which extends under the grid is negligible. The inherent springiness of the metal presses the grid close to the underside of the platform, and keeps the grid from shifting when placed crosswise.

To reduce object-film distance to a minimum a simple cassette-lifting mechanism (Fig. 8) is employed. Heavy rods (about 3/16 inch diameter) (a pair each for cassettes No. 2 and 3) bent into long flat "U" shapes (*23*) are used to bring the cassettes in turn against the Lysholm grid for serial exposure. The first cassette is in this position at the outset, since it lies on top of the stack of cassettes. When the first cassette has been exposed and pulled out of the tunnel, the one underneath is quickly lifted into the position just vacated, by thrusting forward the handle (*24*). The bent rods extend through the side of the tunnel, terminating in loops which fit into holes at either end of the handle (*25*).

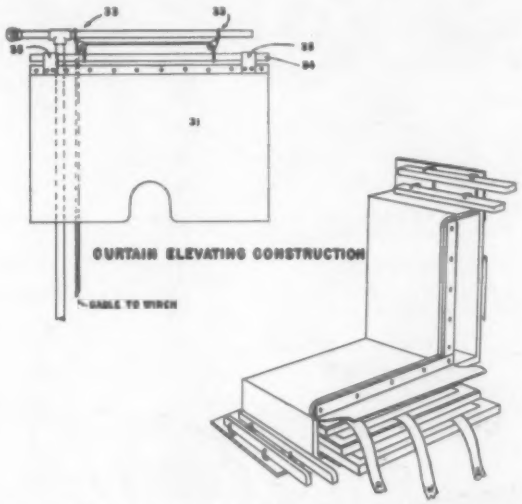

Fig. 7. Diagram to show curtain elevating construction and cassette tunnels.

As the handle is thrust back it causes these loops to turn, exerting a cranking action which rotates the rods inside. The depth of the legs of the "U" shape is such that when the rods are rotated to vertical, they lift the cassette the exact distance required to meet the Lysholm grid. Obviously the rods for cassette No. 2 are bent to a shallower "U" than those for cassette No. 3 (since both cassettes No. 2 and 3 are lifted for the second film of the series). Thus the bends in rods No. 3 will be deeper than those in rod No. 2 by the thickness of one cassette.

This mechanism is duplicated in both cassette tunnels. Covering the entrance to both tunnels are closure flaps (*26*) of heavy lead rubber which prevent the escape of stray radiation. The lead rubber, while flexible enough to "give" to a cassette as it is pulled out, is at the same time springy enough to snap back into protective position after its passage.

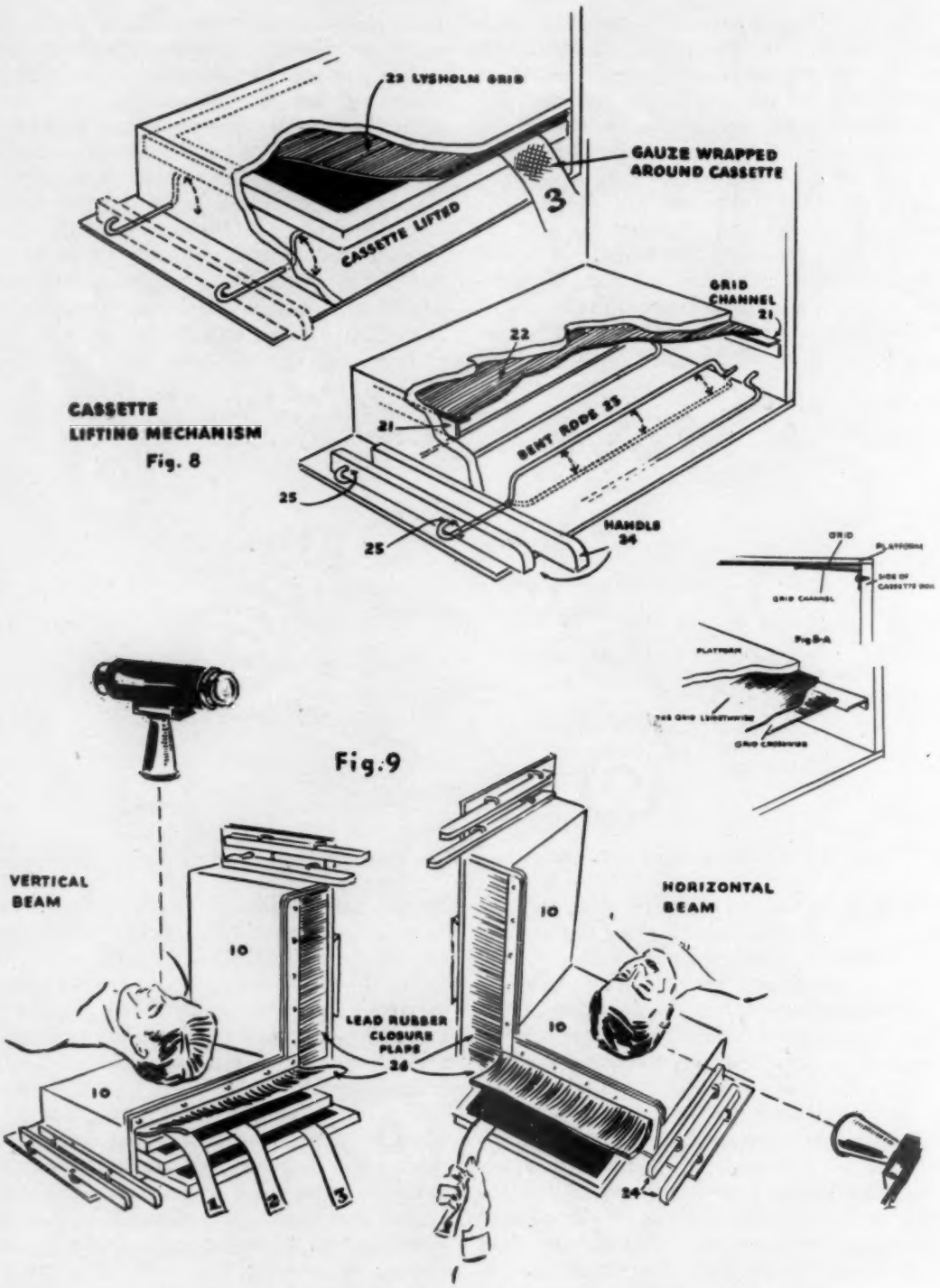

Fig. 8. Diagram to show cassette lifting mechanism.

Fig. 8*A*. Diagrams to show channel construction for positioning of grid lengthwise or crosswise.

Fig. 9. Diagram to show use of cassette tunnels for anteroposterior and lateral angiography.

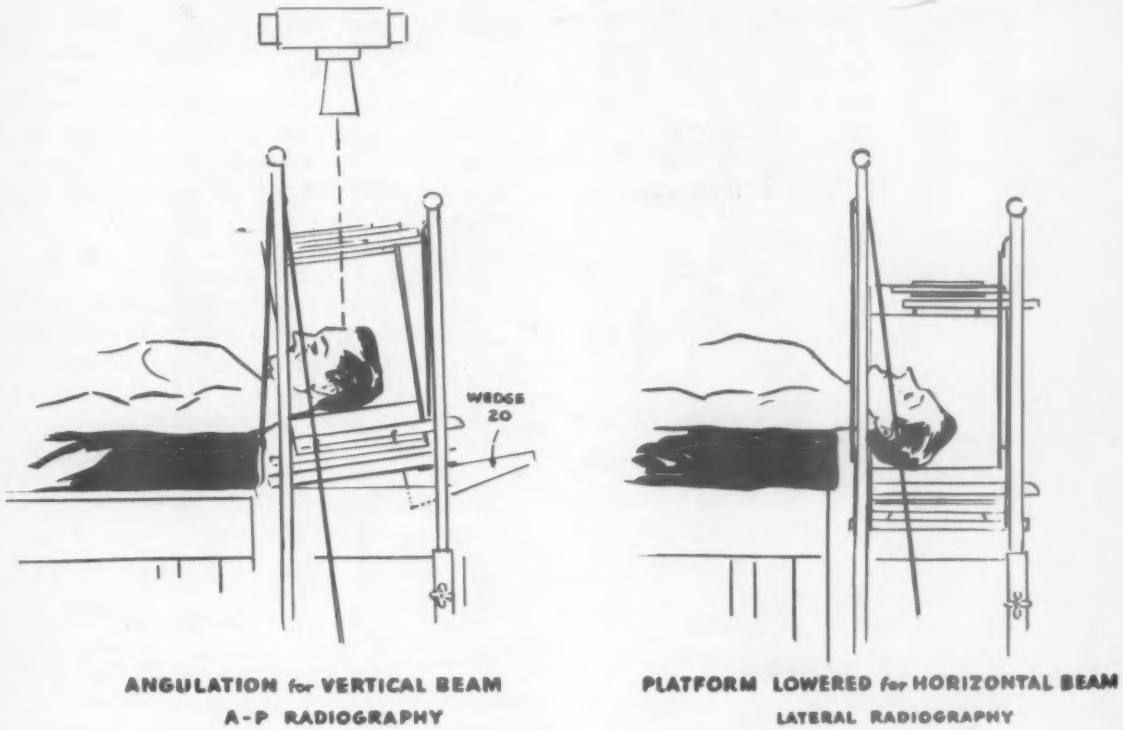

FIG. 10. Diagram to show head angulating and head lowering mechanisms for anteroposterior and lateral angiography.

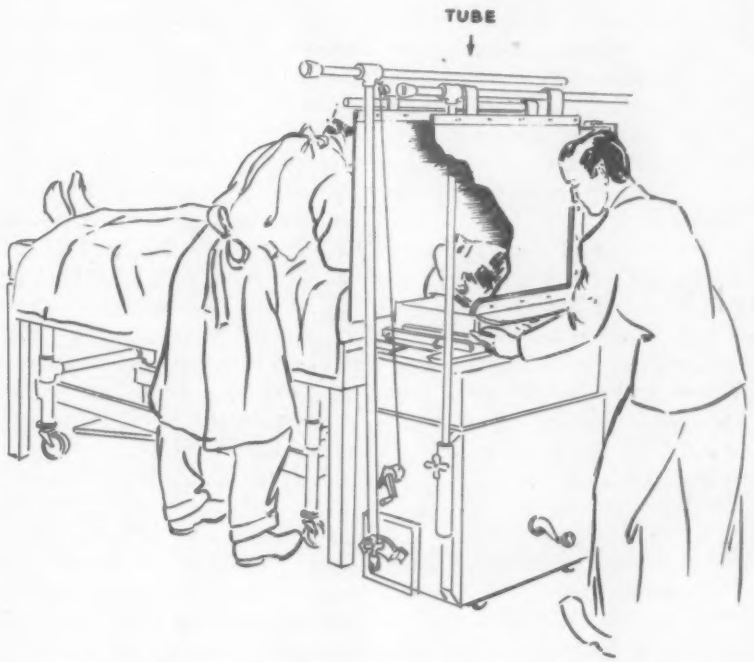

FIG. 11. Diagram to show lead aprons in place for protection of neurosurgeon and roentgen technicians.

Protection for the Neurosurgeon. The neurosurgeon working at the front and the technician at the back of the serialograph are both protected against radiation by lead rubber curtains suspended adjustably as follows: a vertical mast (*27*) constructed of iron pipe is pivoted at its lower end (*28*) so that it may be swung

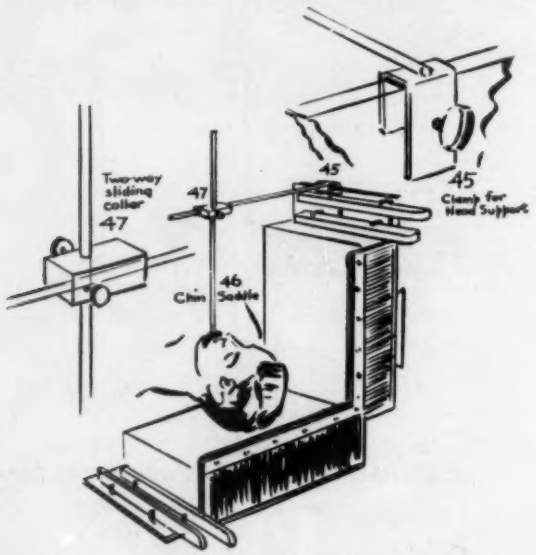

Fig. 12. Diagram to show clamp for head support.

radially and locked by knob (*29*) at any angulation within the range of movement permitted within the bracket (*30*). The purpose of this movement is to permit the curtain (*31*) to be moved forward or backward to accommodate differences in the conformation of patients. Passing through a "T" elbow at the top of the column is a horizontal member (*32*) which serves to support at either end a pair of pulleys (*33*). Hung from these pulleys is a rod (*34*). Hung from the rod by metal hooks (*35*) is the lead rubber curtain (*31*) which can be lowered or raised as its supporting steel strand cable is wound or unwound on winch (*36*) rotated by a worm-and-gear mechanism. This flexibility of adjustment permits the utmost precision in the placement of the curtain, viz.:

a. Positioning in the vertical plane by swinging mast (*27*) forward or backward.
b. Lateral shift by sliding the curtain left or right on its supporting hooks on the horizontal arm.
c. Final adjustment to the neck of the patient by the winch (*36*). This last adjustment can be made with such delicacy that the arched neck opening can be brought beneath the patient's chin without contaminating the operating field. Recommended procedure (once the apparatus has been set up and the patient's head is resting on the platform) is to move the curtain upward out of the zone of operation so that the neurosurgeon may have a clear field for his work until everything is in order: the position of the needle checked, and the saline or glucose injected to demonstrate the patency of the needle. When these final checks have been completed, the curtain is lowered until it touches, but does not press against the patient's neck. A lead-rubber sheet is then placed over the upper part of the patient's chest so that it overlaps the curtain. In this manner the neurosurgeon receives optimum protection against scattered radiation, a consideration of great importance in view of the proximity in which he must perforce operate.

Protection for the Technician. A second vertical mast (*37*) is mounted so that it can slide within the larger diameter pipe sleeve (*38*) and be locked by set-screw knob (*30*) at any necessary height. A horizontal arm (*40*) passes through the "T" at its upper end. Suspended by bent hooks (*41*) from this cross bar is a lead rubber curtain (*42*). A ledge (*43*) follows the back edges of both cassette tunnels. It serves to support the lead rubber closure flaps (*26*) which cover the entrance to the tunnels. These flaps extend to the top of the ledges, and are fastened to them by screwed-on strips of wood. By adjusting the height of the horizontal arm (*40*) and moving the curtain laterally, the curtain can be positioned so that it fits snugly behind the ledges (*43*) at both bottom and side, providing an overlap which prevents radiation from reaching the technician who works behind it. Further confinement of radiation is achieved by a lead-lined rayboard (*44*) which is hung on the back of the cassette tunnel in the vertical position.

Vertical or Lateral Beam Roentgenography (Fig. 9, 10). A unique advantage of the serialograph described is the facility provided for either vertical or lateral roentgenography without having to move the patient. Since both legs of the device are identical, it can be shifted to either position (*a*) or (*b*) by sliding the tongue board at the back of either leg into the mating channel on the supporting board. Tilting action provided by wedge (*20*) together with the rising

and falling head platform (*10*) permits the head to be placed at any desired elevation and angulation.

A chin support to maintain immobility (Fig. 12) clamps on top of the lead-lined rayboard (*44*). This device is a simple "U" clamp with a thumb screw (*45*). A horizontal rod extends horizontally from this clamp. A two-way sliding collar (*47*) has holes drilled through it at right angles so that the horizontal rod and the vertical rod may be adjusted independently for position by set screws which lock travel in either direction. At the lower end of the vertical rod is a saddle (*46*) which embraces the patient's chin, maintaining the head immobile during roentgenography.

TECHNIQUE OF SERIALOGRAPHIC ANGIOGRAPHY

At Montefiore Hospital intracranial angiography is carried out almost routinely in those cases in which an intracranial operative lesion is diagnosed clinically, whenever the lesion can be lateralized clinically, by electroencephalography or by routine stereoscopic roentgenography of the skull. The following procedure is employed:

The ambulatory patient is placed in the supine position on the serialographic table. If non-ambulatory he is brought to the roentgen department on the stretcher portion of the serialographic device.

This stretcher portion is wheeled into position under the cross-arm of the roentgen tube stand and is converted into a fixed roentgen table by lowering the table to the floor, after withdrawal of the supporting cleats as previously described. At this point, the patient's head is momentarily supported by an attendant, the head-rest of the stretcher is pushed into place under the stretcher top and the serialographic section of the device is wheeled into place and is locked securely to the table, thus forming a single immobile roentgenographic unit.

The relative positions of the roentgen tube stand, cross-arm and roentgen tube and serialograph are then so adjusted that it will be possible later to roentgenograph the skull of the patient both laterally and anteroposteriorly without changing the position of the patient's head, by merely swinging the tube and cross-arm from one position to the other and by sliding the serialograph cassette tunnels into proper position.

After the serialograph has been placed in position, the protective leaded aprons are hung from the cross-arms (Fig. 4, *40* and *32*) and are adjusted for protection of the technician and neurosurgeon.

The winch (Fig. 4, *36*) for lowering the protective leaded apron over the neck of the patient is a very useful mechanism since it permits very slow lowering of the leaded apron and exact adjustment to the neck of the patient.

Following completion of these arrangements, the neurosurgeon proceeds with the percutaneous injection of the diodrast.

In those cases in which the attempt at percutaneous injection is unsuccessful the serialographic section of the device is disconnected, the head-rest is pulled out into position, the table is again transformed into a stretcher by the mechanism previously described and the patient is wheeled into the operating room for incision and fixation of the needle in the artery. The patient is then brought back to the roentgen department where the stretcher is again lowered to the floor and attached to the serialographic portion of the device.

When everything is in readiness, one roentgen technician is assigned to the serialograph and an assisting technician to the control stand of the roentgen machine. The neurosurgeon begins the injection of 10–15 cc. of 35 per cent diodrast and after the injection of about 6–8 cc. orders the first exposure to be made, in the meantime continuing the injection.

Immediately after the first exposure has been completed, the technician at the serialograph removes the exposed No. 1 cassette, pushes the lever which elevates the No. 2 cassette into position and orders the second exposure to be made. He then immediately removes the No. 2 cassette, pushes the No. 3 cassette in place and orders

the third exposure to be made. The exposures are made at predetermined time intervals. A clock with a 15 inch face and its easily read second hand, hung where it can easily be seen by the technician, is used for this purpose.

With a little practice the three exposures can be completed in about four to five seconds, resulting in the production of an arteriogram, a phlebogram of the first phase and a phlebogram of the second phase.

The patient remains in position, his head supported by the chin clamp (Fig. 12).

The technician immediately begins development of the first three films. The surgeon in the meantime disconnects the syringe from the needle and instills saline or glucose solution from time to time to keep the blood from clotting in the needle.

If the first injection has been successful and the desired roentgenograms have been obtained, the technician shifts the position of the tube for purposes of stereoscopic visualization of the cranium and its vasculature. The neurosurgeon injects another 10–15 cc. of the diodrast and the second series of the stereoscopic set of roentgenograms is obtained, during the three phases of the intracranial circulation.

In most instances the two sets of stereoscopic films can be paired successfully for stereoscopic visualization of the intracranial vessels. These stereoscopic views are extremely useful in the study of these vessels.

When for any reason satisfactory roentgenograms are not obtained either after the first or second injection, the injection may be safely repeated a third or fourth time for the required supplementary series of roentgenograms.

After completion of the angiographic examination in the lateral position, the roentgen tube is swung into position for the anteroposterior views, the position of the serialograph is adjusted, the patient's head is gently elevated and tilted forward by the cranking device (Fig. 3, *11*) and wedge (Fig. 10, *20*) and the procedure is repeated, as above described for the lateral angiography in the lateral position.

At Montefiore Hospital, intracranial angiography has been successfully carried out in 148 cases to date. In some cases as much as 60–70 cc. of the 35 per cent diodrast has been injected intra-arterially in six or seven 10 cc. doses. In the entire series, no untoward effects have been observed. The serialographic device, above described, has been found to be highly satisfactory and the procedure has been established and accepted as one yielding invaluable information with a minimum of risk and discomfort to the patient.

The writer wishes to express his gratitude to the Picker X-ray Corporation for help in the preparation of the illustrations and working drawings of the serialographic device.

The writer also wishes to express his thanks to Mr. Arthur Muscat of the maintenance department of Montefiore Hospital for his kind cooperation and aid in the construction of the serialographic device.*

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* Copies of the working drawings of the serialograph will be sent by the writer to any physician or hospital on request.

Vol. 61, No. 3

UNRESOLVED PNEUMONIA

AN EXCLUSION DIAGNOSIS*

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THE roentgenologist plays a prominent role in the diagnosis, classification and follow up study of pneumonia. Upon him rests the responsibility of interpreting the changes which take place within the pneumonic lung—particularly after the acute prostrating illness is past.

Many times when the patient is about to go home a chest film reveals persistent shadows in the lung. These shadows are by no means easy to evaluate correctly but are more significant than is generally realized. Too frequently we are tempted to take the course of least resistance, use the convenient label of "unresolved pneumonia" and forget the matter in view of the apparent well being of the patient. This, I believe, is a grave mistake for by so doing we fail to emphasize to the clinicians that recovery is incomplete and that treatment and study should be continued until those shadows disappear or are thoroughly understood. Most chronic pulmonary diseases causing disability today can be traced back to a previous pneumonia of some sort from which the patient failed to recover fully. It is evident that accurate diagnosis and intelligently directed complete treatment of the pneumonia patients being cared for today will materially lower the incidence of chronic pulmonary disease in the future.

Pneumonia was once a serious disease which merited and received grave consideration and treatment. Today, as a result of modern drugs and antibiotics, the mortality rate has been so reduced that once the diagnosis has been made, the disease is apt to receive summary or even perfunctory treatment. Due to the "masking effect" of antibiotics, the symptoms and clinical signs of pneumonia soon disappear and unless physicians remember that the pathological and physiological changes within the lung do not always keep pace with the clinical improvement, we shall continue to court danger. There is a strong tendency to get pneumonia patients "up and out" earlier and to forget that there are sequelae which can and do lead to chronic pulmonary invalidism. When pneumonia-like shadows persist longer than usual, the term "unresolved pneumonia" is temptingly handy and once this soothing appellation is applied, there is little expectation of careful anatomical diagnosis or physiological treatment.

It is the purpose of this paper: (*a*) to emphasize that study and treatment of pneumonias should be continued until complete resolution, or until persisting shadows are adequately explained; (*b*) to outline a planned course for study of post-pneumonic shadows in the chest; (*c*) to discourage the indiscriminate use of the term "unresolved pneumonia."

THE PNEUMONIAS IN GENERAL

There are generally considered to be a great many different kinds of pneumonia. This is no doubt true, but to the radiologist the common denominator of all pneumonias is the exudate. The diagnosis of pneumonia rests upon the finding of exudate and recovery from pneumonia is dependent upon the resolution of the exudate.

For practical purposes we can classify all forms of pneumonia depending upon the situation of the exudate as follows:

a. Alveolar
   Exudate primarily in alveoli
   Example—lobar pneumonia

* The opinions expressed in this paper are based upon cases studied at Walter Reed General Hospital, Washington, D. C., the 98th General Hospital in England, and at the Geisinger Memorial Hospital in Danville, Pennsylvania.

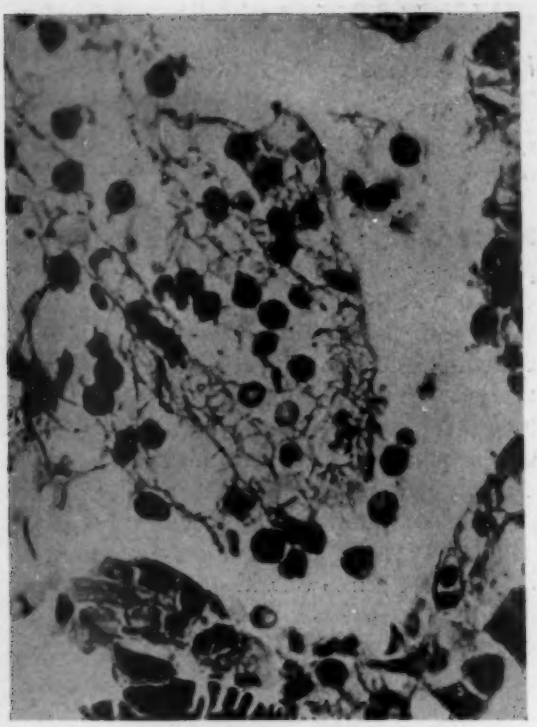

Fig. 1. Fibrinous exudate in early lobar pneumonia × 600. (Courtesy, Boyd, Wm. Pathology of Internal Diseases. Lea & Febiger, Philadelphia.)

b. Interstitial
   Exudate in alveolar walls and bronchial connective tissue
   Example—primary atypical or virus pneumonias
c. Mixed
   Exudate in connective tissue, bronchi and alveoli
   Example—any or all bronchopneumonias

RESOLUTION IN PNEUMONIA

Resolution of inflammatory exudate in pneumonia is a complex and amazingly efficient process which in the great majority of cases results in complete return of the diseased lung to normal.[9] In alveolar lobar pneumonia it is unusual to have incomplete resolution, as the exudate is acted upon by enzymes liberated by the pneumococci and by the leukocytes with resultant complete clearing of the lung.[2] In the interstitial and mixed pneumonias, the smaller bronchi and bronchioles are often seriously injured and even though the exudate is liquefied it is not completely evacuated.[4,8] In such pneumonias, there is occlusion of bronchi and bronchioles and re-aeration may be spotty and incomplete. Areas of atelectasis develop and when this occurs the positive-negative intra-alveolar pressure changes cannot take place, the normal inspiratory elongation and dilatation of the bronchi is absent, and the sweeping action of the cilia is ineffectual.[13]

Occasionally it happens that the mechanism of resolution (or lysis of the pneumonia) is incomplete or too slow, and the process of organization begins.[9] This consists of the proliferation of fibroblasts and vascular buds which extend from the alveolar septa and grow into the undissolved exudate in a criss cross manner. In lobar pneumonia these cells grow on the latticework of fibrin already present (see Fig. 1). The process is similar to organization seen elsewhere in the body and it is indeed remarkable that it does not occur more frequently in the lung. If the organization proceeds to completeness, it eventuates in dense fibrosis of the bronchioles and of the

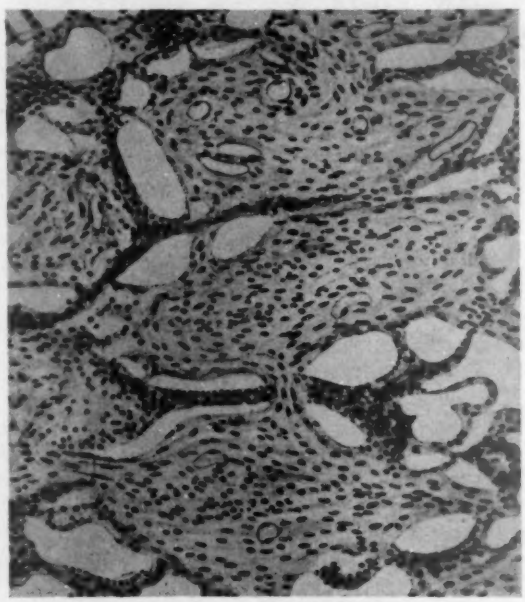

Fig. 2. Carnification of lung in five week old pneumonia. Vascular connective tissue filling alveoli and extending through septa. Medium magnification. (Courtesy Reimann's translation of Kaufmann's Pathology.[10])

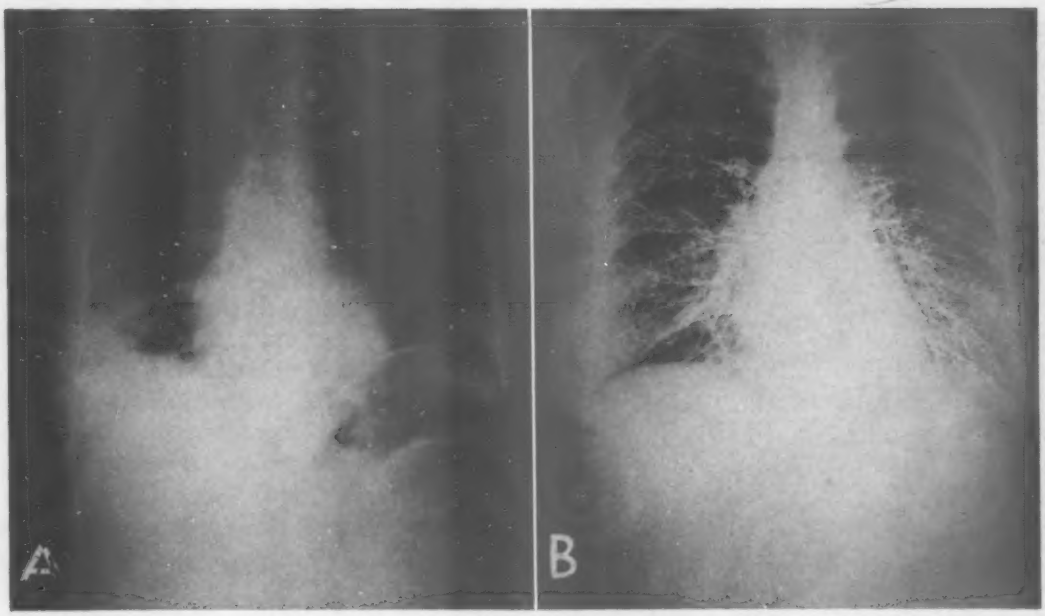

Fig. 3. Case 1. *A*, film after clinical recovery from pneumonia of the mixed variety involving the right upper and middle lobes. These shadows (arrows) were attributed to atelectasis or unresolved pneumonia. *B*, bronchogram after first bronchoscopic aspiration revealed clearing of the upper lobe shadows, improvement in the middle lobe, and occlusion of several branch bronchi in the right middle lobe. All the abnormal shadows disappeared within the next few days.

alveoli and fibers extend through the tiny pore canaliculi, giving the lung a grossly "fleshy" appearance known as "carnified lung" (see Fig. 2). Animal experiments have shown that an excess of serum favors non-resolution and organization.[12]

The process of organization may be assumed to begin in every pneumonia which is slow in resolving and, as it is but a natural and physiological process of reparation, is not to be particularly feared. Any true unresolved pneumonia will of necessity eventuate in fibrosis or organization of the exudate. Such organization may be legitimately called unresolved pneumonia. It is erroneous to use this term if one refers to atelectasis or bronchiectasis, even though the roentgenological and clinical manifestations may be very similar.

PERSISTING POSTPNEUMONIC SHADOWS

The mere fact that a patient has had pneumonia and shows persisting roentgenographic shadows in the chest does not justify the assumption that the pneumonic exudate has failed to resolve.

A group of British workers[14] found that in a series of 38 cases of so-called "unresolved pneumonia,"

18 had obstructed bronchi produced by mucous plugs
8 had carcinoma of the bronchus
5 had bronchiectasis
2 had intrabronchial foreign bodies.

In this country no such startling series has been reported but my experience leads me to believe that we are not being sufficiently thorough. When the bronchial tree is adequately studied, it is found that very few shadows persisting after pneumonia can be attributed to true unresolved pneumonia. Almost always there is some other explanation, the most common of which is bronchial occlusion.[7]

Case 1. Mrs. H. R., aged thirty-three, had a mixed pneumonia involving the right upper and right middle lobes. The pneumonia cleared promptly in two weeks except for a few residual shadows in the right upper and the right middle lobes. Because of the residual strands of density (attributed to atelectasis) (Fig. 3*A*) she was bronchoscoped. Bronchoscopy disclosed noth-

FIG. 4. Case II. Chest film of male L. S., aged twenty. The triangular shadow at the right cardiophrenic angle persisted two weeks after a pneumonic episode. Bronchoscopy revealed a timothy head in the right lower lobe bronchus.

ing significant on inspection, but the aspirating tip extracted thick mucus from both of the involved bronchi. The upper lobe shadows disappeared but the middle lobe shadows remained.

A few days later a bronchogram revealed occlusion of the terminal bronchi in the lateral bronchopulmonary segment of the right middle lobe and persisting shadows in the adjacent lung parenchyma (Fig. 3*B*). Obviously the parenchymal densities are produced by atelectasis. When the bronchial obstruction was removed the lung became entirely normal in appearance.

CASE II. We considered L.S., male, aged twenty (Fig. 4) to have pneumonia in the right lower lobe. He improved but the shadows persisted for two weeks and he was therefore bronchoscoped. Our bronchoscopist removed a timothy head from the right lower lobe bronchus and his "unresolved pneumonia" promptly resolved. The patient denied knowledge of the foreign body.

BRONCHIECTASIS

The literature concerning bronchiectasis emphasizes the etiological importance of previous respiratory tract infections, particularly those in childhood.[13] It is well accepted that persistent bronchopulmonary infection will produce bronchiectasis. Atelectasis and stagnation of bronchial secretions tend to promote chronicity of pulmonary infections, and must therefore be considered as important predisposing factors and as such must be combatted from the onset.

Grier[5] points out that many cases of supposed atypical pneumonia are in reality unrecognized bronchiectasis. Conversely in 40 cases of proved bronchiectasis, he found that 67 per cent had been misdiagnosed as atypical pneumonia.

Blades and Dugan[3] describe temporary cylindrical dilatations of the bronchi during and after atypical pneumonia. These can best be studied by serial bronchograms.

Karpel, Waggoner and McCown[8] studied 500 cases of primary atypical pneumonia and found bronchiectasis to be the most common complication.

Large autopsy series show an over-all incidence of 4 per cent of bronchiectasis, practically all of which can be traced back to a previous pulmonary disease which lingered or recurred. Kay[11] reports in detail on 20 soldiers who had no symptoms or roentgenographic findings prior to atypical pneumonia, but had symptoms and bronchograms typical of bronchiectasis immediately thereafter. The obvious way to prevent such sequelae is to minimize atelectasis, lingering pneumonias and bronchopulmonary necrosis at the time of and immediately following the acute pneumonic episode.

PULMONARY DISEASE MAY ANTEDATE THE PNEUMONIA

In the analysis of postpneumonic shadows it is well to remember that the patient may have had bronchiectasis, tuberculosis, bronchial stenosis, pulmonary infarct, atelectasis or other disease of the lung or bronchial tree before the onset of acute symptoms which brought him to the physician. Many cases diagnosed as atypical pneumonia by the clinician and the roentgenologist are really bronchiectasis with a

new flare up. Often such patients will volunteer the information that they have "had pneumonia five or ten times." Ogilvie[15] has found this history in 36.8 per cent of his large series of cases of bronchiectasis. Obviously patients with such pre-existing disease will not have a clear chest film after the pneumonia because the chest was not clear before it. Surely this should not be referred to as "unresolved pneumonia."

Too frequently one hears the statement made that "some pneumonias take two or three months to clear up." I do not think any pneumonia should be permitted to persist longer than three weeks without a complete tracheobronchial study and I believe that any so-called pneumonia which does linger for months is not pneumonia, but atelectasis, bronchiectasis, or pulmonary fibrosis.

RADIOLOGIST'S RESPONSIBILITY

In the vast majority of cases it is the radiologist who finds the persisting shadows of density in the lung after the acute episode is over. It is the radiologist who evaluates and interprets the shadow and whose function it is to guide the clinician in his attempts to cure the patient. Atelectasis should be allowed to remain no longer than it takes to diagnose it. Bronchoscopic aspiration is an invaluable therapeutic procedure and is becoming more and more accessible.

Example. A roentgenological shadow of density in the lung found after clinical recovery from one of the pneumonias is a diagnostic challenge to the radiologist, and is of immense clinical importance to the future well being of the patient. Let us consider, for example, a roughly triangular shadow of increased density in the lower right lung field which has persisted following a pneumonia. The chest film (Fig. 3*A*) will suffice as an example. As we see this shadow on the conventional posteroanterior view we should be honest enough and wary enough to recognize at once that we cannot make a diagnosis. We must first know a great deal more than can be known from the posteroanterior survey film. A shadow of this kind should bring to one's mind a differential diagnosis list including the following:

Pleural thickening
Interlobar collection
Marginal atelectasis
Pulmonary infarct
Bronchial occlusion
Bronchiectasis
Bronchiolar occlusion
Unresolved alveolar exudate
(Unresolved pneumonia)

Other studies must be made to differentiate the location and nature of the shadow in question. The order in which these are carried out is somewhat a matter of preference. We prefer the following steps in the following order and offer them here for the consideration of others.

Step I. Roentgenoscopic study and oblique and lateral films will tell us whether the shadow is flat or has depth, whether it is near or in a fissure, and in which portion of which lobe it lies. This step should rule in or out:

Pleural thickening
Interlobar collection
Marginal atelectasis
Pulmonary infarct

Step II. Step II is bronchoscopy, which at our clinic is readily available and freely utilized. For didactic purposes we speak of bronchoscopy as functioning in two ways:

1. Diagnostic bronchoscopy, which rules in or out foreign body, tumor, stenosis, or gross inflammatory disease of the primary and secondary bronchi. Particular attention is focused on the orifice of the involved lobe as previously determined by the radiologist and a culture is taken of any material coming from this bronchus. (This is tested in the laboratory for penicillin, sulfa, and streptomycin sensitivity.)

2. Therapeutic bronchoscopy, which consists of introduction of the aspirating tip into the smaller branches of the suspected bronchus during forced coughing. The purpose of this is to extract the tenacious and viscid secretions which the patient has not been able to raise from the

abnormal bronchial tree. Epinephrine spray is used to shrink the mucosa and reduce the muscle spasm. Such dilatation of the bronchi and aspiration has repeatedly resulted in prompt aeration of the involved lobe and disappearance of the roentgenographic shadows in our clinic. (Postural drainage and ammonium chloride have been worth while adjuncts.) (Aminophyllin intravenously has been found to relax bronchospasm.)

If bronchoscopy is negative it excludes the trachea and larger bronchi as the sites of disease.

Step III. The third step is bronchography. This is not done if the diagnosis is clear from bronchoscopy and bronchiectasis is not suspected. Bronchography is done however if:

1. The endoscopist has not found the answer.
2. Bronchiectasis is suspected.
3. It is desired to obtain a pictorial record of the tracheobronchial tree.
4. In certain cases for academic interest.

Bronchography affords three dimensional visualization of the small bronchi and bronchioles not accessible to the bronchoscope and permits accurate localization of bronchial obstruction, bronchiectasis, or other abnormalities.

Only when it has been shown that the large and small bronchi are patent and normal are we justified in attributing the roentgen shadow to exudate or fibrosis in the pulmonary parenchyma. The diagnosis of unresolved pneumonia should be made only by exclusion.

CONCLUSIONS

Study and care of pneumonia patients should be continued until all pulmonary shadows have disappeared or are proved to be due to fibrosis.

True unresolved pneumonia is rare while bronchiectasis, atelectasis and intrabronchial diseases are more common.

Unresolved pneumonic exudate is situated in the alveoli and interstitial tissues where it may eventuate in fibrosis.

Unresolved pneumonia is a diagnosis which can be established only after disease of the tracheobronchial tree has been excluded.

The indiscriminate and inaccurate use of the term "unresolved pneumonia" is disparaged.

A methodical course of procedure for such exclusion studies is suggested.

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Vol. 61, No. 3

UNFUSED OSSIFICATION CENTERS ASSOCIATED WITH PAIN IN THE ADULT*

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THERE are many primary and secondary centers of ossification throughout the body which may remain unfused, but in only two locations have we found them actually to be associated with pain: (1) the tarsal scaphoid (navicular), and (2) the tibial tubercle. These two osseous structures are sites of predilection for aseptic necroses which will likewise cause pain due to local nutritional disturbances of the epiphyses. In this discussion, however, we are considering the accessory scaphoid bone and the tibial tubercle as unfused ossification centers associated with pain, particularly in the adult, without a history of a so-called osteochondritis† in the developmental period.

A. ACCESSORY SCAPHOID (SYN.: EXTRA SCAPHOID, DIVIDED NAVICULAR, OS TIBIALE EXTERNUM, OR PREHALLUX)

Historical. The accessory scaphoid or divided navicular, as it is commonly called in the literature, has been described by anatomists for many years, but it was not until the discovery of the roentgen rays that a more thorough investigation of this bone was instigated. It is the largest of the supernumerary structures appearing in human feet, and probably the most troublesome. Pfitzner[8] states that it can be demonstrated in 10 per cent of all individuals; Schindler and Gnagi[18] in a series of 200 unselected individuals found it in 8 persons, or 4 per cent; whereas, in a series of 5,000 patients, we found it in only 5 per cent. Many physicians, however, are still unaware of the fact that this bone does exist, and may be associated with pain and tenderness, and for this reason it is frequently mistaken for a fracture of the tuberosity of the scaphoid. This fact was observed by one of us (D. W.) in the military service, where accessory scaphoids were erroneously interpreted as fractures and treated as such. The pain, however, persisted despite the use of plaster casts. These patients were eventually referred to a regional hospital, where operative removal of the extra bone resulted in a cure.

This bone was first described by Bauhin in 1605. Pfitzner[8] in 1896 gave a voluminous account of it and named it the os tibiale externum. Dwight[8] in 1907 likewise referred to it as the tibiale externum but divided it into two types: (1) the true tibiale externum, a true part of the skeleton, which actually represents the isolated tubercle of the scaphoid, and (2) the tibiale externum, a small rudimentary ossicle farther out in the tendon of the tibialis posticus muscle. This supernumerary bone arises in antiquity and according to Kidner[14] is seen in many of the lower mammals to a greater or lesser degree of development and with greater or lesser functional importance. Froehlich speaks of it as "atavistic debris." Monahan[16] suggests that it is a direct evolutionary descendent of the prehallux or sixth toe in such lower vertebrates as the echidna and opossum. This extra bone has, therefore, also been designated the prehallux, because it is directly homologous, both in position and form, with the prehallux in these lower animals. Geist[10] has reported 27 cases under the term, "disturbance of the accessory scaphoid," and in 1915 was the first surgeon to perform an operative removal of this bone.

Embryology and Anatomy. The accessory scaphoid exists as a preformed cartilage in the second month of fetal life. This bone,

† Most observers, notably Caffey, now prefer the use of the term osteochondrosis, since the condition is often seen asymptomatically and without accompanying inflammation.

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however, does not manifest itself roentgenographically until the age of nine and more usually eleven. Dwight states that he has seen it as a separate cartilage at birth and as separate bone at two years of age, but this is unusual.

The extra scaphoid is usually well outlined and of uniform density and represents the largest regular anomaly of the tarsals. The true tibiale externum, as it has been referred to by Dwight, develops as an ossification center of the scaphoid, which instead of uniting normally with the body

Fig. 1. Accessory scaphoids in three adults, associated with pain, tenderness and a prominence along the mesial aspect of the foot.

of that bone, makes an independent development as an extra ossicle. It represents a portion of the scaphoid bone which envelops the internal aspect of the head of the astragalus by 1 to 2 centimeters, and is separated from the scaphoid proper by a transverse fissure. It is generally closely connected with the tuberosity of the scaphoid by fibrocartilage or fibrous tissue but, on the other hand, it may be quite free and have no close connection with the scaphoid. It is situated at the insertion of tendon of the tibialis posticus muscle and is firmly fixed in position by the tendon. The tendon of the tibialis posticus divides into two chief divisions, a deep and a superficial. The deep portion becomes attached chiefly to the tubercle of the scaphoid bone, and, usually in part, also to the first cuneiform. When the accessory scaphoid is present, the tendon, on its way to its final insertion, attaches to it, instead of the under surface of the scaphoid tubercle. The smaller pea-sized, rudimentary tibiale externum is the one commonly seen, and is located farther out in the tendon of the tibialis posticus, being usually some distance from the regular scaphoid. It is the true tibiale externum, however, that we are concerned with, as this is the osseous element which is responsible for the pain, tenderness and prominence along the mesial portion of the foot. Since the true tibiale externum shares the duties of the regular one piece scaphoid, it is better described as the accessory scaphoid or divided navicular. There is a tendency for the independent accessory scaphoid to be symmetrical, although it is rarely found the same size in both feet. On one side, one sometimes encounters a well formed extra ossicle, while on the other side, a small rudimentary ossicle may be encountered. It is also to be remembered that not all the extra scaphoids are separated from the major mass of the bone. That is, there may be a distinct osseous element on one foot, while the opposite foot may show a consolidated element in the form of an enlarged or hooked scaphoid.

A full account of these many variations in size and shape have been elaborated on by Mouchet and Moutier.[17]

Pathologic Physiology. The following theories and explanations have been propounded as to the underlying pathology responsible for the onset of symptoms.

1. Traumatic Synovitis of the Posterior Tibial Tendon. The deep division of the tendon of the tibialis posticus muscle is attached chiefly to the tubercle of the scaphoid bone and, usually in part, also to the first cuneiform. When the accessory scaphoid is present, the tendon on its way to its final insertion attaches to it, instead of the under surface of the scaphoid tubercle. In some of these cases a dissection showed that the accessory bone, when present, was completely enclosed in the conical expansion of the tendon. As a result of this ab-

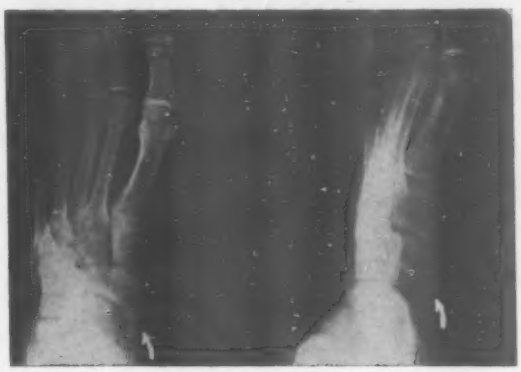

Fig. 2. A painful accessory scaphoid in a male, aged thirty, clearly shown in both the anteroposterior and oblique views.

normal relationship, the tendon sheath of the tibialis posticus may be readily irritated by pressure, and thereby produce a traumatic synovitis analogous to Quervain's disorder of the wrist.[7]

2. Faulty Mechanics of the Foot. When the accessory scaphoid is of any considerable size, the tendon of the tibialis posticus may be displaced upward and inward by its attachment to the inner side of the bone. As a result, the line of its pull is changed. Instead of pulling directly upward, as it does when attached to the under surface of the scaphoid, it is forced to pull backward and inward at an angle, and becomes an adductor instead of a supinator. In all such cases, full adduction of the foot brings the prominent inner end of the bone into close and sometimes painful relation with the internal malleolus.[14]

3. Traumatic Arthritis. According to

Fig. 3. An unusual location of bilateral painful accessory scaphoids in a male, aged thirty-six.

Geist,[10] a true articulation may be present with a hyaline cartilage, synovial membrane and ligaments between it and the scaphoid. Following a mild injury, an arthralgia may develop from a degenerative arthritis in a malplaced and adventitious joint. Geist and Robertson report that they have actually observed this in 2 cases.

4. Bursitis. According to some of the anatomists, notably Gray and Piersol, there sometimes exists a bursa between the tendon of the tibialis posticus and the bone immediately in front of it. Because of mechanical irritation, the extra scaphoid initiates a bursitis.[10]

5. Secondary Inflammatory Changes in the Surrounding Soft Tissues. Growing from the inner border of the scaphoid and directed downward and somewhat backward, the accessory scaphoid produces a

prominence on the surface of the foot. This extra bone is thus in a position to receive much of the weight of the body, and is accordingly a constant point of pressure. The surrounding tissues subsequently become irritated and swollen, and cause considerable pain.[1]

Symptoms and Signs. Pain is the outstanding and only symptom in the region of the tubercle of the scaphoid. We know that this pain is significant because it is relieved by operative removal of this supernumerary bone. On examination, marked tenderness can be elicited at the same site, and occasionally, redness and swelling may be noted.

Fig. 4. Different forms of accessory scaphoid which may be mistaken for a fracture. The line of demarcation from the scaphoid has the features of an epiphyseal line. These accessory bones were discovered accidentally and were unaccompanied by symptoms. Dwight refers to this type of supernumerary bone as the true tibiale externum.

Differential Diagnosis.

a. Fracture. This accessory bone is frequently mistaken for a fracture of the tuberosity of the scaphoid for the following reasons:

1. The presence of a prominence on the inner side of the foot.
2. Pain and tenderness may occasionally accompany the presence of this ossicle.
3. The fact that the finding may be unilateral, having no homolateral counterpart.
4. A history of injury.

It is to be noted, however, that an isolated fracture of the scaphoid is quite rare. If it does occur, the fragments are at first sharply defined, and then later, one of the fragments will become more dense apparently because of a disturbed circulation. The opposing surface of the other fragment at the same time will become sclerotic. On the other hand, the presence of a smooth contour and texture suggests the absence of a fracture. Finally, the fact that the finding is usually bilateral will help in the differential diagnosis. For this reason, one should always have a roentgenogram of the opposite side for comparison.

b. Osteochondritis of the Scaphoid Bone (Köhler's disease). This condition, characterized by a retardation of osseous de-

velopment of the scaphoid bone, is also associated with pain and tenderness. The ossification center for this bone remains small and irregular in outline. This condition, however, occurs in young children from five to ten years of age, while an inflammatory accessory scaphoid usually does not appear before the twelfth year.

Treatment. In the cases that we have observed, the treatment has been for the most part surgical. However, conservative measures can be tried at first, and depending on the severity of the pain, bed rest with hot application, the use of braces, or even a plaster cast may be employed. If no relief is obtained, then operative procedures are recommended. The operation as described by Schindler and Gnagi[18] should include an excision of the accessory scaphoid and a placement of the abnormally attached posterior tibial tendon to a better mechanical position. After excision, the foot is firmly bandaged and motion is restricted for ten days. These two procedures, in the majority of cases, provided a symptomatic cure.

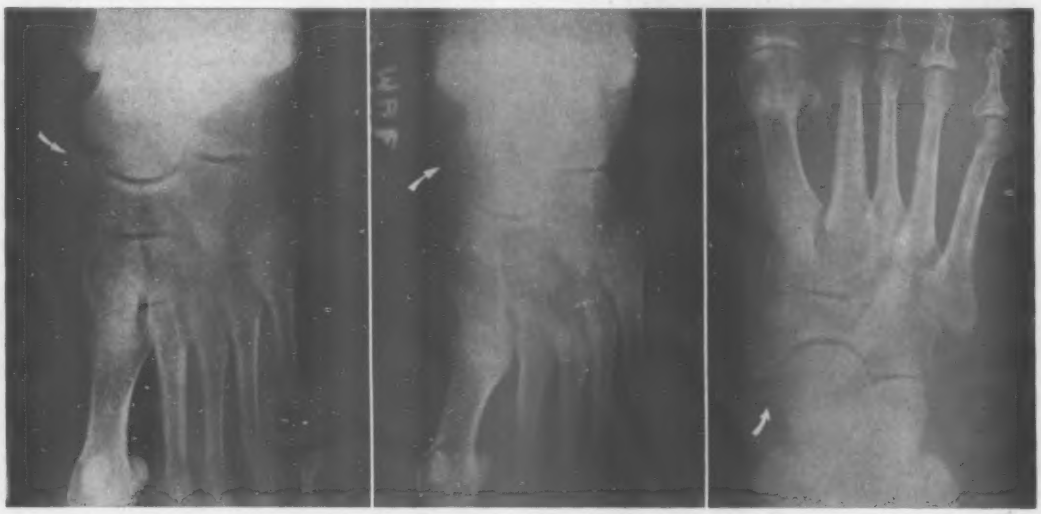

FIG. 5. Smaller pea-sized, rudimentary accessory scaphoids, which are located farther out in the tendon of the tibialis posticus, being some distance from the regular scaphoid. This smaller ossicle does not produce any pain and is rarely mistaken for a fracture. Dwight refers to it as the tibiale externum in contrast to the true tibiale externum described in Figure 4.

B. THE TIBIAL TUBERCLE

Embryology and Anatomy. The proximal extremity of the tibia consists of two condyles which become confluent anteriorly and form a somewhat flattened surface of triangular outline, the apex of which is known as the tubercle of the tibia. The tubercle is divided into two parts: the upper part is rounded and smooth; the lower part is rough and receives the insertion of the ligamentum patellae.

The anatomical development of the tibial tubercle proceeds in the following manner: The proximal tibial epiphysis ossifies at or just before birth. At some time in early childhood, the tibial tubercle develops from a tongue of cartilage growing from the proximal tibial epiphysis over the anterior aspect of the tibial metaphysis. In this cartilaginous tongue an ossification center develops, which is usually single, although it may be multiple. This ossification center makes its appearance in most instances at twelve years of age. Fusion of the tibial tubercle apophysis with the proximal tibial epiphysis occurs at about fourteen years of

age. Roentgenographically, this is represented by a beak-like projection, which hangs downward in front of the diaphysis and constitutes the so-called tongue-shaped process of the proximal tibial epiphysis. However, if this ossification is incomplete, a separate osseous nucleus makes its appearance at the distal extremity of the lingula. This has been termed the anterior epiphysis which subsequently unites with the tongue-shaped process. Fusion of the lingula with the shaft of the tibia usually occurs at the age of eighteen years but may be delayed until the age of nineteen.

Numerous variations in the development and fusion of the tibial tubercle may occur:

1. The apophysis for the tibial tubercle may develop on both sides of the same individual, but in different directions and to a different extent on the two sides.

2. The ossification centers may appear earlier on one side than on the other.

3. An additional second osseous nucleus may appear distal to the lingula so that the tubercle may present a different picture on the two sides.

4. There may be a disturbance in the fusion of the tibial tubercle, either with the proximal tibial epiphysis or with the adjacent tibial diaphysis.[15]

Pathologic Physiology. From the foregoing description of the embryological development of the tibial tubercle, it is apparent that this segment of bone in subject to anatomical variations in size, shape, manner of growth, and degree of fusion to the adjacent shaft of the tibia. As a result of these variations, the ligamentum patellae will have a different length of insertion and will present an alteration in its anatomical relationship to the tibial tubercle. The patellar ligament is a continuation of the central portion of the quadriceps tendon, some of the fibers of which are prolonged over the front of the patella into the ligament. It is an extremely strong flat band, which is attached above to the lower border of the patella and below, it is inserted into the lower part of the tubercle and the upper part of the crest of the tibia. The ligament thus overlies the apophysis, which in turn is in contact with the apophyseal plate, which finally is in contact with the tibial metaphysis. Roentgenologically, the unfused tibial tubercle manifests itself as a loose fragment of bone in the ligamentum patellae, and physiologically, it serves to transfer the pull of the quadriceps muscle through the semifibrous raphe between the fragment and the tibia. The bones in such an anatomical arrangement constitute weak points. In addition, the attachment of the patellar ligament at its insertion is mechanically inadequate. The principal point of application of forces, although well supported by less taut collateral structures, is extremely small, while the functioning quadriceps muscle is the largest in the body.[6] The pull of the muscle is always in an upward direction, in the plane of the anterior tibial crest. This direction persists, in its relation to the tibia, for any angle of flexion at the knee. This is so because the patellar ligament is located well below the axis of the hinge-like motion of the knee joint and because the infrapatellar fat acts as a block to prevent the ligament from imposing directly on the tibial epiphysis when the quadriceps acts on the flexed knee.[21]

The assumption is that, because of the weak bones existing in these structures, there is a constant danger of disruption. It may thus be explained why excessive or abnormal traction on the patellar ligament will produce pain in the region of the tibial tubercle. This traction on the patellar ligament may occur without interfering with the blood supply to the tibial apophysis. This would therefore preclude the possibility of an aseptic necrosis or Osgood-Schlatter disease.

Roentgen Findings. The tibial tubercle may normally show an unusual degree of variability in size, shape and texture in different persons and in the same person on the two sides. Consequently, the roentgen appearance will be that of an independent tibial tubercle, which may be smooth or irregular in contour, normal or sclerotic in

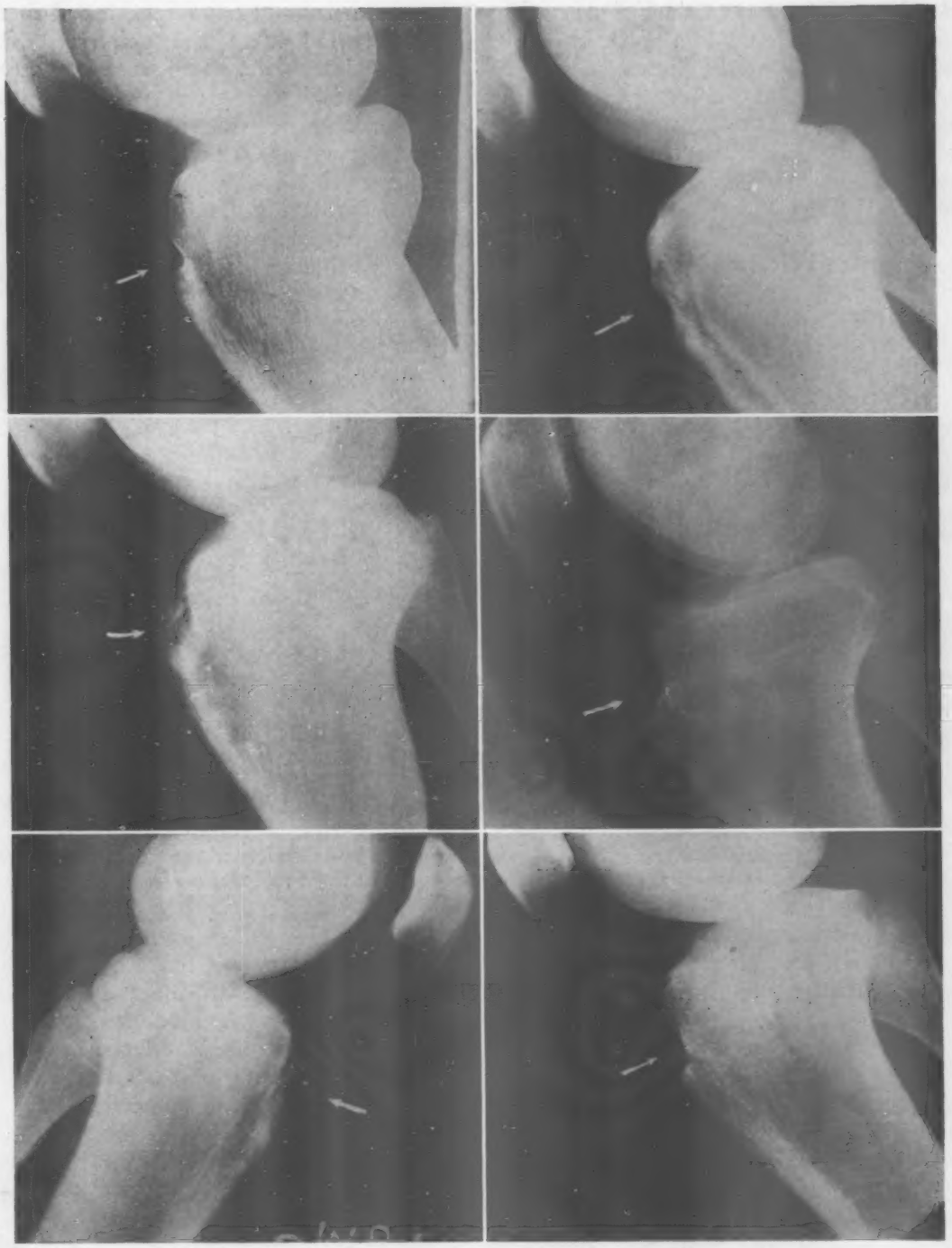

FIG. 6. Varieties of unfused tibial tubercles in adults, accompanied by pain, and without a history of an osteochondritis during the developmental period. These independent tubercles are smooth in contour, of uniform density, and show no evidence of fragmentation or abnormal calcifications in the patellar tendon.

density and may even show signs of fragmentation. A routine film of the knee joint may reveal any of the above findings despite the fact that the patient presents no symptoms. On the other hand, the same roentgen appearance may be present in an individual who complains of pain in the region of the tibial tubercle, independent of an aseptic necrosis. The roentgenological findings are therefore a result of some defect in development in which there has never been a fusion of the tubercle with the diaphysis.

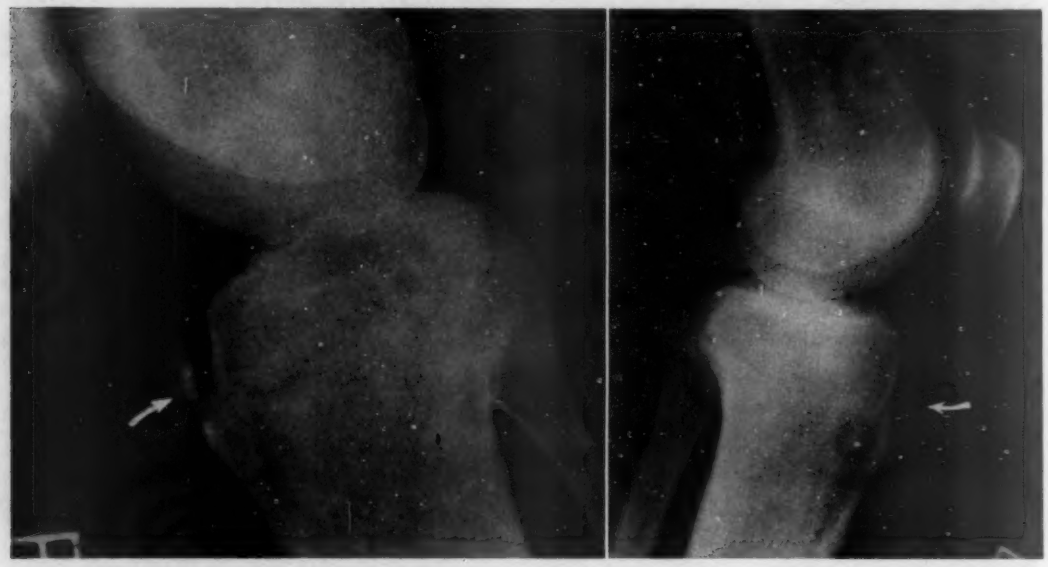

Fig 7. Varieties of unfused tibial tubercles in adults, accompanied by pain, and without a history of an osteochondritis during the developmental period. These independent tibial tubercles are irregular in contour, sclerotic in density and show evidence of fragmentation.

Differential Diagnosis.

Osteochondritis of the Tibial Tubercle (Osgood-Schlatter Disease). This condition, which is essentially an aseptic necrosis of the tibial tubercle, is differentiated simply by the fact that it is found in children between the ages of ten and sixteen prior to the union of the epiphysis. And, unless we can assume that an unfused tibial tubercle and an osteochondritis can occur independently or simultaneously, there is little need for the condition to be considered in the differential diagnosis, since the cases that we are discussing have been found in the adult period after the epiphyses have closed. Heretofore, there have been some observers who have erroneously considered the condition we describe, as included in the category of Osgood-Schlatter disease. However, by definition, this disease can only occur during the growth period.

The actual pathological changes in Osgood-Schlatter disease are still debatable. The explanation that the process is one of trophic degeneration and necrosis is the prevalent one. As a result of exercise, tension of the ligament on the epiphysis may cause a tearing or partial separation of the tibial tubercle. This tension may decrease the blood supply to the epiphysis, which is normally received from the patellar tendon route, until bony union occurs. The end result is a necrosis of the tibial tubercle with marked osteolysis and subsequent bone proliferation. On the other hand, other observers are of the opinion that traumatic fragmentation of the tibial tubercle is responsible for the clinical and roentgen findings.

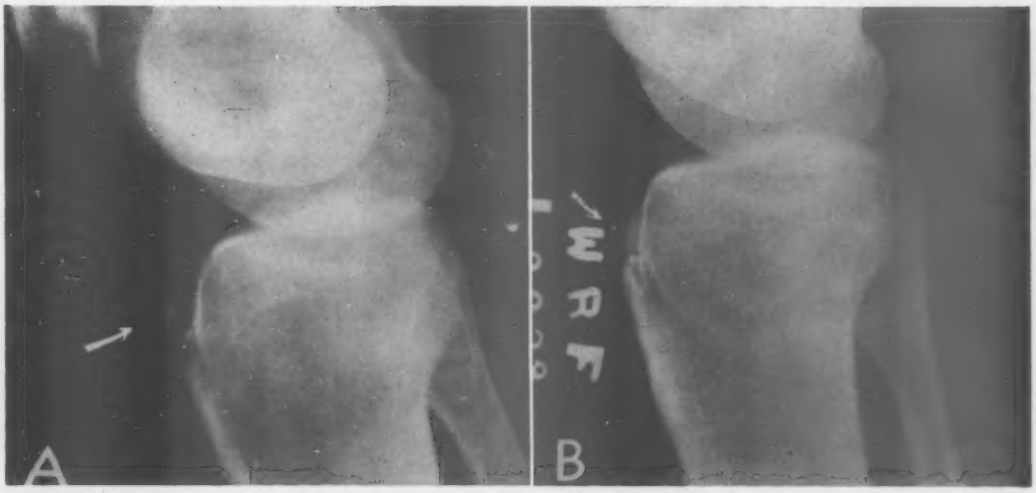

FIG. 8. Bilateral unfused tibial tubercles in a female, aged twenty-two. *A*, the independent tibial tubercle on the right side is fragmented at its inferior margin and is accompanied by pain. *B*, the left side was taken for comparison. There was no history of an osteochondritis during the developmental period.

The clinical symptoms are those of local pain, tenderness, swelling and limitation of motion, chiefly extension. Any or all of these findings may be absent except the soft tissue swelling in the region of the tibial tubercle, which is a fairly constant finding. It is to be noted that the swelling is entirely confined to the patellar tendon, the interval between the tendon and skin showing no swelling.

The roentgenological findings vary according to the nature and extent of the injury. An enlargement or thickening of the patellar ligament is the earliest and most constant finding in this disease. This is followed by secondary changes in the form of irregular spicules of bone, which may be seen radiating from the anterior surface of the tubercle and subsequently extend into

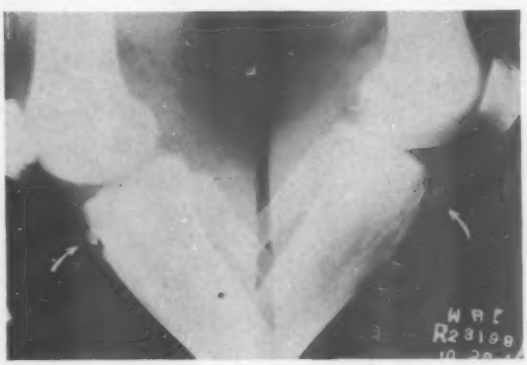

FIG. 9. Bilateral unfused tibial tubercles in a male, aged twenty-five. The independent tibial tubercle on the right side is irregular in contour, sclerotic in density, fragmented and associated with pain. The left side was taken for comparison. There was no history of an osteochondritis during the developmental period.

FIG. 10. Bilateral unfused tibial tubercles in a male, aged twenty-two. The independent tibial tubercles on both sides are irregular in contour, sclerotic in density and fragmented. Pain was present on the left side, while the right side was taken for comparison. Again, there was no history of an osteochondritis during the developmental period.

the patellar tendon. Actual islands of bone may be seen within the tendon, representing either ossifications in the tendon or avulsion fragments from the tubercle. The tibial tubercle itself will present an alteration in texture, an irregularity in contour, or the tubercle may be broken up into small or large fragments with the displacement of the fragments from the shaft.

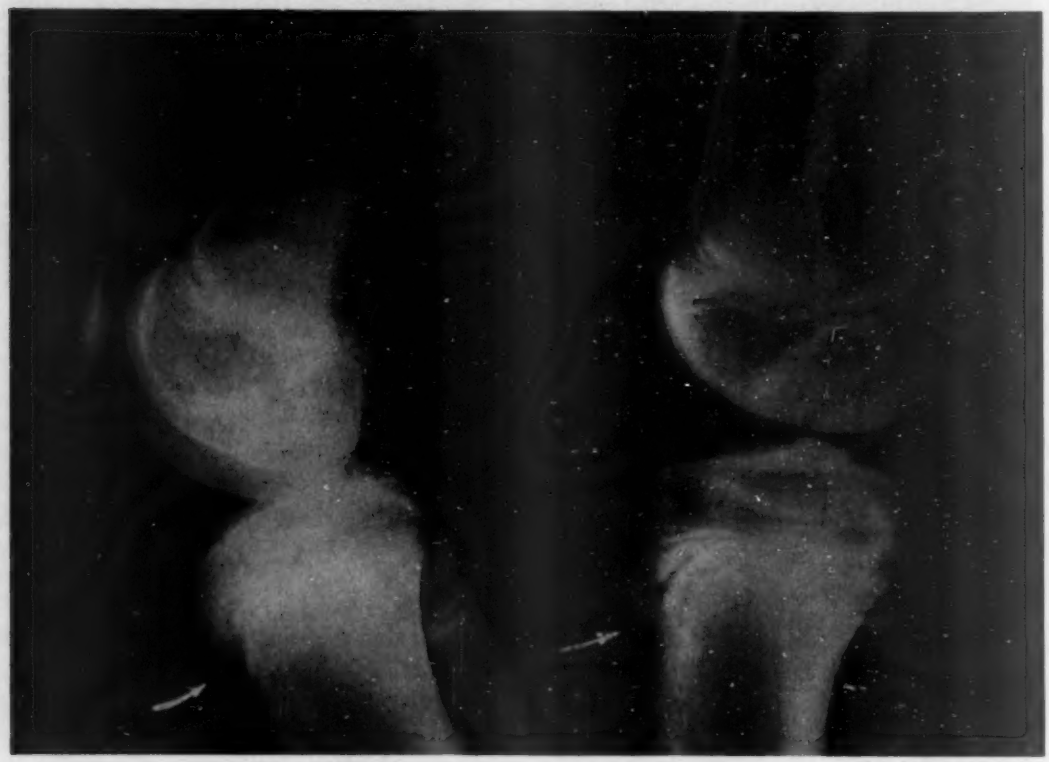

FIG. 11. Bilateral Osgood-Schlatter disease in a boy thirteen years old. The independent tibial tubercles present an alteration in texture and an irregularity in contour. Irregular spicules of bone can be seen radiating from the anterior surface into the patellar tendon. A thickening of the patellar ligament was present on the original roentgenogram. These findings were accompanied by localized pain, tenderness, swelling and limitation of motion.

Treatment. Cross strapping with adhesive or elastic bandage together with physiologic rest invariably resulted in a cure.

No operative form of treatment was necessary in any of the cases which we have observed.

DISCUSSION

This paper has attempted to present the clinical and pathological findings associated with the unfused ossification centers in the regions of the tarsal scaphoid (navicular) and the tibial tubercle. Too often the radiologist overlooks the fact that the accessory scaphoid and tibial tubercle may be associated with clinical symptoms, independent of an osteochondritis, and fails even to mention their presence when describing a roentgenogram. On the other hand, because of the presence of clinical symptoms, the accessory scaphoid will be erroneously diagnosed as a fracture while the unfused tibial tubercle will be called Osgood-Schlatter disease.

In our discussion of the accessory scaphoid, numerous theories have been mentioned regarding the underlying pathology responsible for the symptoms. The abnormal relationship of the insertion of the

Fig. 12. Bilateral Osgood-Schlatter disease in a boy twelve years old. The independent tibial tubercles show an alteration in texture, an irregularity in contour and suggestive evidence of fragmentation. Calcific spicules can be seen on the anterior surface extending into the patellar tendon. A thickening of the patellar ligament was present on the original roentgenogram. These findings were accompanied by localized pain, tenderness, swelling and limitation of motion.

tendon of the tibialis posticus to the extra scaphoid bone is, in our opinion, the most likely explanation. When the accessory scaphoid is present, the tendon in the development of its final insertion attaches to it, instead of the under surface of the tuber-

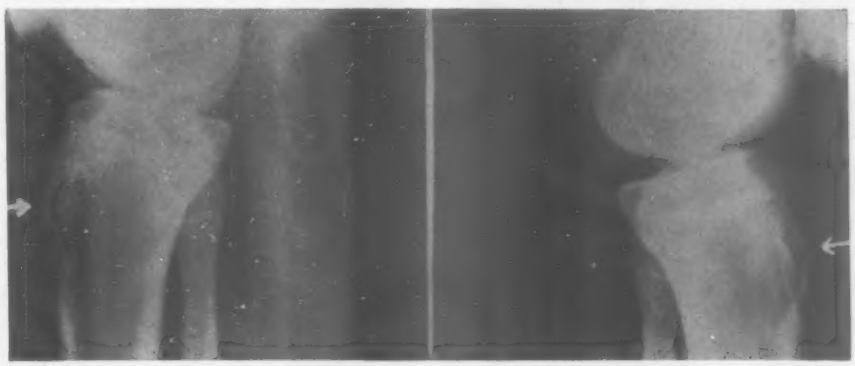

Fig. 13. Bilateral Osgood-Schlatter disease in a boy fourteen years old. The independent tibial tubercles again present an alteration in texture and an irregularity in contour. Actual islands of bone can be seen in the patellar tendon. The roentgen findings were accompanied by the usual symptoms. The bilateral occurrence of this condition is found in approximately 14 per cent of cases.

cle. The tendon sheath of the tibialis posticus is thus subjected to irritation by pressure, resulting in a traumatic synovitis. There may also be a disturbance in mechanics of the foot, so that instead of pulling directly upward as it does when attached to the under surface of the scaphoid, it is forced to pull backward and inward at an angle, thus becoming an adductor instead of a supinator. Either of these two elements may come into play and account for the local pain and swelling.

The tibial tubercle is subject to a wide variety of patterns which are too often regarded as pathologic. It is to be borne in mind that an irregularity in contour and density and even the presence of fragmentation in the tibial tubercle may occur in asymptomatic individuals. Similarly, the same roentgen appearance may occur in the presence of pain in the region of the tibial tubercle, independent of an osteochondritis. This observation was quite evident among the soldiers undergoing basic training, which one of us (D. W.) had the opportunity to observe. In most instances the age of the soldier indicated that fusion of the tibial tubercle with the adjacent metaphysis had simply been retarded thereby tending to eliminate the possibility of an osteochondritis as the cause for symptoms. Excessive traction on the patellar ligament accounted for the pain because of the preexisting weak bonds between the tibial tubercle, tibial metaphysis and the patellar ligament. These changes were comparable to the painful swellings at the site of insertion of the posterior tibial tendon into the accessory scaphoid.

In order to establish a diagnosis of an osteochondritis, however, additional and more reliable roentgenological evidence is necessary. This includes a thickening of the patellar ligament at its insertion, the presence of irregular bony spicules extending from the anterior surface of the tubercle into the ligament, the existence of islands of bone in the ligament itself, and finally fragmentation of the tibial tubercle with displacement of the fragments away from the shaft. These roentgenological findings, accompanied by clinical symptoms and signs, tend to favor a diagnosis of Osgood-Schlatter disease, when they are observed in the adolescent child before the age of thirteen to fourteen years.

It may be theoretically possible to have a painful unfused epiphysis in the growing child without roentgen evidence of an osteochondritis, as described above, particularly when trauma has been superimposed. This may, however, lead to the roentgen findings of a typical osteochondritis somewhat later.

SUMMARY

1. The unfused ossification centers in the region of the tarsal scaphoid (navicular) and the tibial tubercle may be associated with pain independent of a so-called osteochondritis.

2. Each bone in the area of involvement is discussed individually and the clinical and pathologic significance is evaluated.

3. Emphasis is placed on the differential diagnosis. The accessory scaphoid must particularly be differentiated from a fracture and the unfused tibial tubercle must be distinguished from Osgood-Schlatter disease, granting that the two conditions can occur alone or simultaneously during the growth period.

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MONOSTOTIC FIBROUS DYSPLASIA*

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MONOSTOTIC fibrous dysplasia is one of the more common bone lesions seen in man. Many instances of this condition have been reported under various titles, the most common of which are "osteitis fibrosa," "osteodystrophia fibrosa" and "fibrocystic disease of bone." In 1938, Lichtenstein[3] suggested the term "fibrous dysplasia" in an effort to clarify the confused terminology referable to multiple bone lesions associated with extraskeletal defects; namely, pigmentation of skin, and precocious puberty in females, first described in this country by McCune and Bruch[6] in 1937. Albright and his associates[1] in the same year described the identical syndrome, classifying the skeletal lesions as "osteitis fibrosa disseminata." In 1942, Lichtenstein and Jaffe[4] extended the term "fibrous dysplasia" to include an identical solitary lesion of bone not associated with extraskeletal anomalies; they used the qualifying term of "monostotic fibrous dysplasia."

As the name implies, fibrous dysplasia is essentially a lesion whereby normal bone is replaced by fibrous tissue in which this basic element is transformed into immature bone trabeculae. Since various stimuli provoke connective tissue proliferation in all parts of the body, the etiology of fibrous dysplasia remains a mystery. It is generally believed, however, that, in the polyostotic forms with the associated extraskeletal anomalies, an endocrine imbalance plays a leading role. Albright, however, in his original communication, was impressed by the widespread distribution of lesions and postulated the theory of a primary disturbance of the central nervous system, possibly in the hypothalamus. Thannhauser's[8] belief of a common factor in von Recklinghausen's neurofibromatosis and fibrous dysplasia has not gained support among other investigators.

The monostotic form of fibrous dysplasia is not easily explained by any of the theories advanced for the polyostotic form; however, Lichtenstein and Jaffe[4] are of the opinion that the monostotic and polyostotic forms merely represent different expressions of the same basic disease. More recently, Schlumberger,[7] in a review of the 67 cases of monostotic fibrous dysplasia studied at the Army Institute of Pathology, was impressed by the apparent role which trauma played in the development of these lesions, and he believes that the monostotic form has nothing in common with the form of polyostotic fibrous dysplasia found in Albright's syndrome. There is, however, little detectable difference between the individual lesions of the polyostotic and monostotic forms.

Monostotic fibrous dysplasia can present a wide variation of manifestations roentgenologically, depending on the distribution of fibrous tissue and the amount and configuration of the metaplastic bone within this basic element. Obviously, such a process will be more progressive during the period of active growth of bone (Fig. 1, *A* and *B*) and less so after adolescence or when the bones have ceased to increase in size and length (Fig. 3, *A* and *B*).

Replacement of fibrous tissue results in an area of diminished density within the involved bone, affecting both the medulla and cortex. This area of diminished density may simulate a cyst or cysts when little or no new bone is present (Fig. 4, *A* and *B*). The lesions may expand the bone, or

* This paper is largely based on cases observed by the authors during their duty at Walter Reed General Hospital, Washington, D. C. Presented at the Forty-eighth Annual Meeting, American Roentgen Ray Society, Atlantic City, N. J., Sept. 16–19, 1947.

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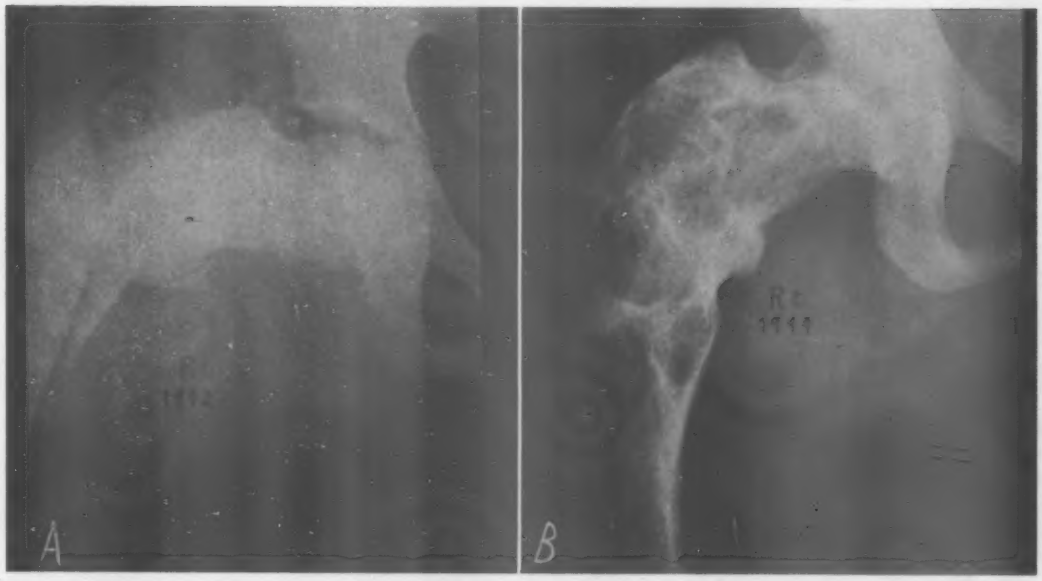

Fig. 1. Case I. Polyostotic fibrous dysplasia. *A*, this case is included for comparison with the other figures and illustrates the appearance of polyostotic fibrous dysplasia in a seven year old white girl. The intertrochanteric area is the site of a pathologic fracture. *B*, the appearance of the lesion of the femur is characteristic. There is, however, a lesion in the inferior ramus of the pubic bone which contains no definite calcification, has sharply defined margins of slightly increased density, and has increased in size over a two year period. The early lesions in polyostotic fibrous dysplasia are less apt to contain calcium and may be sharply defined in contradistinction to the authors' experience with the monostotic form in adults.

may simply replace its structure without alteration in size or configuration.

The following points are of aid in diagnosis:

(1) The formation of innumerable, superimposed, bone trabeculae produces a homogeneous increase in density within many of the apparently cystic areas. This results in what has been termed a "smudged" or "ground glass" appearance[2,6] due to the fact that the bone trabeculae are too small to register as individual shadows on the roentgenogram. Whenever an apparent cyst is of greater

Fig. 2. Case II. Hyperparathyroidism. The bone lesions are indistinguishable from those of fibrous dysplasia except for one additional finding; namely, generalized decalcification of all of the bones. This generalized decalcification of otherwise uninvolved bones is not found in simple fibrous dysplasia and is essential for the roentgen diagnosis of hyperparathyroidism.

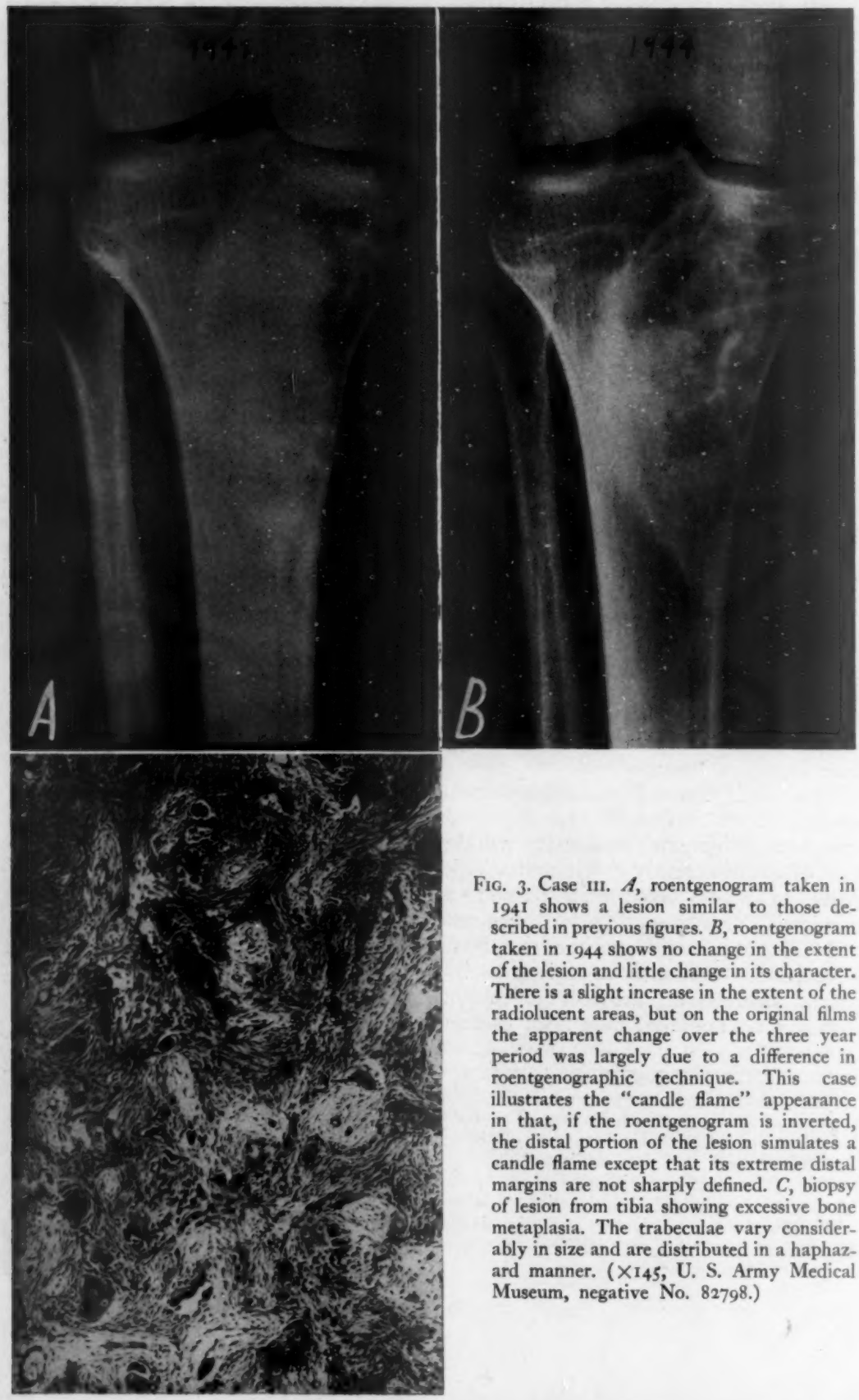

Fig. 3. Case III. *A*, roentgenogram taken in 1941 shows a lesion similar to those described in previous figures. *B*, roentgenogram taken in 1944 shows no change in the extent of the lesion and little change in its character. There is a slight increase in the extent of the radiolucent areas, but on the original films the apparent change over the three year period was largely due to a difference in roentgenographic technique. This case illustrates the "candle flame" appearance in that, if the roentgenogram is inverted, the distal portion of the lesion simulates a candle flame except that its extreme distal margins are not sharply defined. *C*, biopsy of lesion from tibia showing excessive bone metaplasia. The trabeculae vary considerably in size and are distributed in a haphazard manner. (×145, U. S. Army Medical Museum, negative No. 82798.)

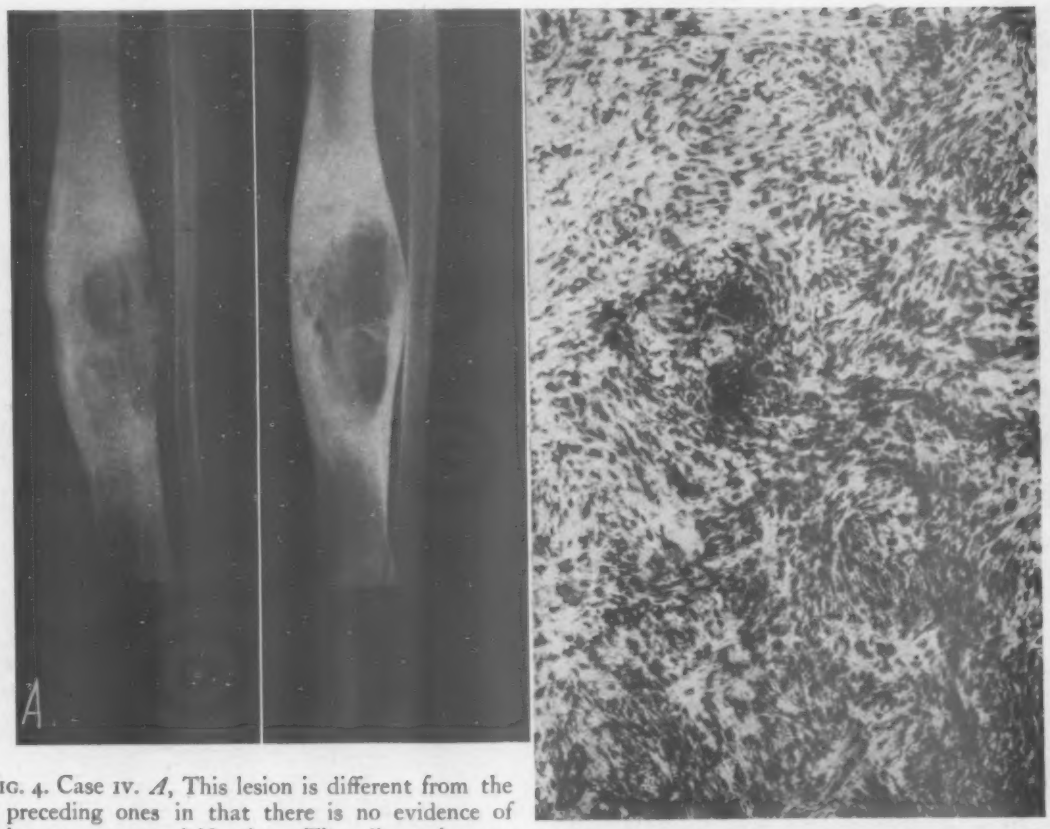

Fig. 4. Case IV. *A*, This lesion is different from the preceding ones in that there is no evidence of homogeneous calcification. The diagnosis was based on lack of definition of the distal margin of the lesion and a portion of the proximal margin, plus the heavy, coarse, irregular columns in the anterior wall of the lesion. *B*, section prepared from radiolucent area in tibia showing absence of bone metaplasia. The stroma here is typical of that seen in fibrous dysplasia. In other sections bone formation was quite evident. (×175, U. S. Army Medical Museum, negative No. 82797.)

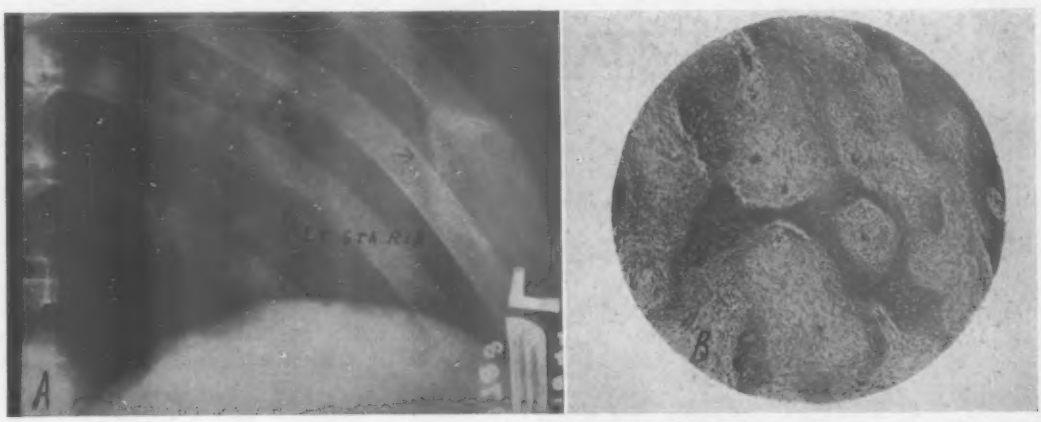

Fig. 5. Case V. *A*, roentgenogram shows a fusiform expanding lesion with margins of increased density. This lesion appears cystic but its central portion is too dense to be explained on the basis of fluid, indicating that it contains radiopaque material. Its cortical margins are smooth, but the margins extending proximally and distally in the rib are poorly defined and shade off into normal bone. Note the "smudged" appearance of the central calcification. *B*, metaplastic bone forming from a dense fibrous stroma. Here the trabeculae approach the architectural pattern of normal bone. (×100, Army Medical School, negative No. 3865-A23.)

Fig. 6. Case vi. This lesion is similar to that shown in Figure 5, *A* and *B* but contains much more calcium as evidenced by the rather marked increase in density throughout the lesion. The degree of calcification varies considerably. The cortex is intact.

Fig. 7. Case vii. This lesion has the characteristics described in the previous figures with the addition of a wide variation in density of the various areas. Obviously, the amount of calcification or ossification found within a section of the lesion will vary according to the site of biopsy.

Fig. 8. Case viii. Roentgenogram shows an extensive multilocular cyst-like lesion with heavily, densely calcified, curving columns traversing it and expansion of the cortex anteriorly. In this latter location there is the frequently seen homogeneous increase in density. The inferior and superior margins of the lesion shade off into normal bone.

density than if it were filled with fluid, it probably represents fibrous dysplasia (Fig. 5*A* and 6).

(2) In many lesions the calcification is dense and irregular, particularly along the periphery of an apparent cyst (Fig. 7). In some instances the lesions assume a multilocular appearance produced by coarse, curving columns of bone (Fig. 9, *A* and *B*).

servations of others. The involved bone is replaced by a firm, rubbery, gray to yellow-white tissue easily identified as fibrous in character (Fig. 10*B*). Whereas occasionally there are small cysts, these are usually not of significant size or number to confuse the roentgenologic picture. This abnormal fibrous tissue consistently has a gritty sensation as a result of the innumerable, newly

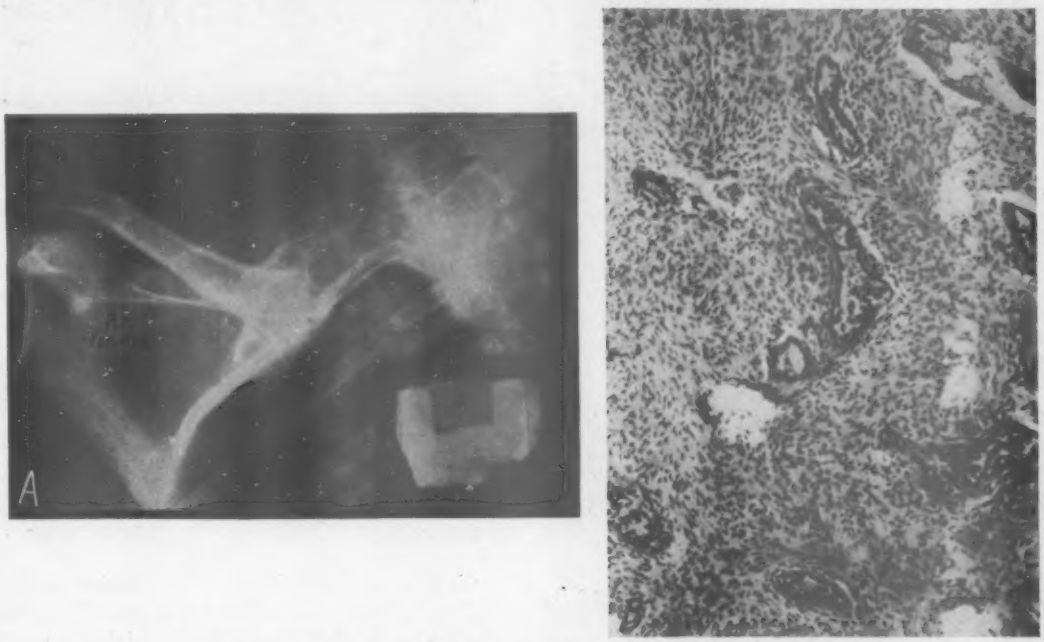

Fig. 9. Case IX. *A*, practically the entire rib is replaced by an expanding "soap bubble" lesion. Note the homogeneous density within the apparently cystic areas. The insert is a specimen roentgenogram of a fragment from the rib. A rectangle has been removed from the fibrous tissue portion of the lesion and replaced with a paraffin block of the same thickness. Note the difference in density between the paraffin and the fibrous tissue due to innumerable immature bone trabeculae which are too small to register as individual shadows on the film. *B*, calcified metaplastic bone in dense fibrous stroma. This section prepared from a wedge represented in Figure 10, *A* and *B*. (×175, U. S. Army Medical Museum, negative No. 82792.)

(3) The increase in density at the periphery of an apparent cyst may extend varying distances into the adjacent bone where it usually shades off into normal bone without a perceptible line of demarcation. This "shading off" is frequently tongue-like in configuration and has been compared to the appearance of a candle flame (Fig. 3*B*). It is one of the more constant characteristics of the lesion.

The gross pathologic picture which was available to us in our study of monostotic fibrous dysplasia coincides with the ob-

formed, immature, but calcified, bone trabeculae.

The basic histologic component of the lesion is fibrous tissue which typically has a dense, fasciculated arrangement, often forming whorls (Fig. 4*B*). The more striking microscopic findings, however, are the irregularly distributed, immature spicules of bone which invariably show varying degrees of calcification (Fig. 3*C*, 5*B* and 9*B*). The degree of this latter change accounts for one of the more consistent important roentgenologic findings. It is un-

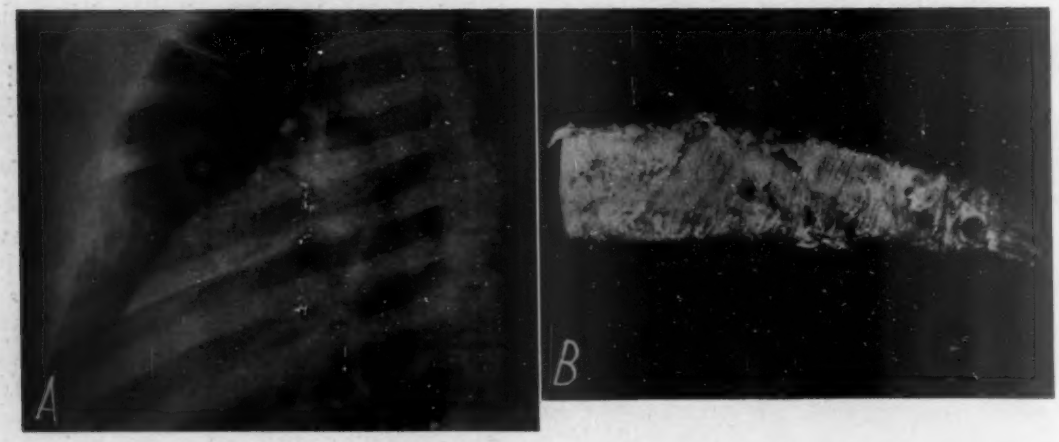

Fig. 10. Case x. *A*, the entire rib is replaced and destroyed by the lesion which is similar in character to that shown in Figure 3, *A* and *B* except that the cortex is also destroyed in places and there is less calcification within the fibrous tissue. In that portion of the rib which is projected clear of the lung, however, there is evidence of a small amount of homogeneous calcium deposit. Here again, the proximal margin of the lesion is not clearly defined. *B*, longitudinal section of seventh rib. The bone is completely replaced by dense, rubbery, gritty fibrous tissue. The cortex is generally destroyed.

usual to find the metaplastic bone more evident at or near the periphery of the lesions, which accounts for the frequent finding in the roentgenogram of a sharp inner margin of increased density (Fig. 4*A*).

Fig. 11. Case xi. The margins of this lesion are sharply defined except for its distal aspect as seen in the lateral view where the lesion shades off into normal bone. This lesion also extends beyond the normal limit of bone laterally. Differential diagnosis between fibrous dysplasia and enchondroma is impossible in this instance because of the sharply defined margins of the major portion of the lesion.

The following cases serve to illustrate the roentgenologic and pathologic changes. As described clinically, the lesions were asymptomatic unless they produced a palpable swelling or were the site of pathologic fractures.

Case i. *Polyostotic Fibrous Dysplasia.*

A white girl, aged seven, presented a classical picture of Albright's syndrome with precocious sexual development, pigmented skin areas and bone lesions involving practically every bone in the body with the exception of the vertebrae. The lesions were predominantly on the right side.

Case ii. *Hyperparathyroidism.*

This is a proved case of hyperparathyroidism with generalized fibrous dysplasia of the bones in which a parathyroid adenoma was removed. (This case was contributed from the Department of Radiology of the Massachusetts General Hospital, Boston, Massachusetts through the courtesy of Dr. Laurence L. Robbins.)

Case iii. *Monostotic Fibrous Dysplasia.*

A white man, aged twenty-seven, prior to induction into the Army sustained an injury to the left knee followed by acute pain and localized swelling. A roentgenogram made immediately following injury revealed an elongated, coarsely trabeculated lesion in the

upper medial aspect of the left tibia. Biopsy of this lesion two months following injury was interpreted as a giant cell tumor. After entering the Army and while on duty overseas the patient complained of pain about the left knee. Following roentgen examination he was sent to Walter Reed General Hospital where the lesion was reclassified as fibrous dysplasia.

Case iv. *Monostotic Fibrous Dysplasia.*
A white man, aged twenty, sustained a blow to the left leg three years before admission. This was followed in about one month by painless swelling which persisted until it was detected on physical examination. A roentgenogram taken at this time led to biopsy and diagnosis.

Case v. *Monostotic Fibrous Dysplasia.*
A white man, aged twenty-two, had indulged in frequent boxing bouts and for a year after induction into the Army complained of aching, sticking pains in the lateral aspect of the left thorax. Shortly before admission to the hospital, while boxing the patient experienced a sudden, sharp exacerbation of pain in the region of the left seventh rib. A roentgenogram taken shortly thereafter revealed an expanding fusiform lesion of the seventh rib. This was later resected and a diagnosis of fibrous dysplasia was made.

Case vi. *Monostotic Fibrous Dysplasia.*
A white man, aged twenty-four, stated that his left leg had been larger than the right as long as he could remember and had gradually increased in size up to four years before admission. It had remained stationary during the past four years. The lesion was asymptomatic except for occasional aching following strenuous exercise.

Case vii. *Monostotic Fibrous Dysplasia.*
A white man, aged twenty-eight, stated he had had a hard swelling of his leg as long as he could remember which had increased in size up to two to three years prior to examination. The swelling was non-tender and was not painful except for occasional aching following strenuous exercise. Biopsy was reported as fibrous dysplasia.

Case viii. *Monostotic Fibrous Dysplasia.*
The patient was a white man, aged twenty-one, in whom the lesion was discovered incidentally on a chest film taken because of pain in the left side.

Fig. 12. Case xii. Paget's disease. This lesion simulates fibrous dysplasia except that it is limited to the cortex and the cortex is thickened. This thickening is demonstrated in the area just above the lateral femoral epicondyle. There are several coarse, vertical strands of bone in the medial portion of the lesion, but these are sharply defined and do not have the "fuzzy," irregular margins of the columns seen in fibrous dysplasia. Paget's disease includes the location of an epiphysis practically universally, but this is not a differential point between Paget's disease and fibrous dysplasia.

Case ix. *Monostotic Fibrous Dysplasia.*
A white man, aged twenty-seven, six years prior to induction into the Army in 1942, sustained trauma to the right shoulder which was immediately painful but became asymptomatic after three weeks of immobilization. While in the Army, symptoms referable to pressure on

Fig. 13. Case xiii. Enchondroma. This lesion simulates fibrous dysplasia perfectly except that its proximal margin is sharply defined, with normal bone immediately adjacent to it.

the right brachial plexus developed and roentgenologic examination revealed a lesion involving the entire first rib.

Case x. *Monostotic Fibrous Dysplasia.*

A white man, aged thirty-seven, had complained of pain over the left anterior chest wall for one and one-half years, aggravated by deep breathing. Two and one-half years previously he had sustained an injury to this area.

Case xi. *Monostotic Fibrous Dysplasia.*

A white female, aged seventeen, noted asymptomatic swelling of extremity just below knee. A biopsy was performed and a diagnosis of fibrous dysplasia made.

Case xii. *Paget's Disease.*

The patient was a white man, aged forty-six. This lesion was discovered because of an injury followed by painful swelling about the right knee which persisted for two weeks, after which a roentgenogram was taken. It was not believed that the lesion had any relation to the clinical changes.

Case xiii. *Enchondroma.*

The patient was a white man, aged thirty-three, with enchondroma of the fifth metacarpal. This lesion was discovered because of an injury which led to roentgenologic examination.

Case xiv. *Enchondroma.*

A white female, aged forty, had a tooth extracted and two weeks later developed pain in the region of the right knee joint. Roentgen examination revealed an asymptomatic lesion in the femur, subsequently biopsied and diagnosis of enchondroma made.

Case xv. *Giant Cell Tumor.*

A white female, aged seventeen, had complained of soreness in the knee and a limp for two years prior to examination. A biopsy of the neck of the left femur revealed a benign giant cell tumor. This lesion did not respond to radiation therapy but continued to increase in extent. The patient subsequently sustained a pathological fracture, and the proximal end of the left femur was resected.

Fig. 14. Case xiv. Enchondroma. Lateral roentgenogram of the femur shows a fairly well defined lesion, except for its proximal and distal aspects where the margins are hazy. Differential diagnosis in this instance between fibrous dysplasia and enchondroma is impossible, as in Case xi.

The most common error in diagnosis is to interpret this lesion as a cyst, and the fact is frequently overlooked that these apparent cysts do not contain fluid but consist of dense tissue with varying degrees of irregular ossification or calcification within the involved area. Other conditions in which the basic pathologic lesion is either similar to or has some of the characteristics of it, such as calcification, and which must be excluded in making the diagnosis, include giant cell tumor, osteochondroma, enchondroma, hyperparathyroidism and Paget's disease. On the other hand, some cases of fibrous dysplasia may simulate malignant bone tumors (Fig. 10, *A* and *B*). Giant cell tumor is more frequently encountered in the long bones, being located in or near the epiphyseal line, and in general shows less trabeculation roentgenographically than the monostotic form of fibrous dysplasia. Osteochondromas are usually more lobulated and expansile in addition to frequently being cystic. Enchondromas may closely simulate fibrous dysplasia (Fig. 13), including the appearance of amorphous calcification. The margins of enchondromas are, however, usually sharp and definite with no alteration in structure or density of the adjacent bone architecture. In addition, enchondromas are much more frequently seen in the small bones, notably the phalanges, than is fibrous dysplasia. Von Recklinghausen's disease of bone is usually polyostotic and associated with demineralization of all the bones to a varying degree. Paget's disease, which is unusual in young adults, shows considerably more osteoblastic activity and is limited to the thickened cortex (Fig. 11), whereas fibrous dysplasia involves both the cortex and medulla.

Fig. 15. Case xv. Giant cell tumor. This lesion involves the head, neck and intertrochanteric portions of the femur with partial destruction of the inferior cortex of the femoral neck. Its margins are sharply defined and the lesion is traversed by a few septa which also have sharp margins. The lesion as a whole, however, contains no calcium, but has rather the density of soft tissue or fluid. This lack of calcification is the most significant differential point, the other being the sharpness of definition of the margins of the lesion. Giant cell tumor involves the location of epiphyseal centers, but this is not a dependable differential point.

SUMMARY

Monostotic fibrous dysplasia presents a widely variable roentgenologic appearance. These variations must, however, of necessity remain within the limits of the gross pathologic changes. The diagnostic aspects are: (1) homogeneous increase in density within apparently cystic areas; (2) an increase in density of the inner margins of the lesion with "fuzzy," irregular columns of bone traversing it; and (3) "shading off" of the peripheral margins of the lesion into the adjacent normal bone. If any one of these changes is observed, the diagnosis of fibrous dysplasia should be suspected; if all of these changes are present, the roentgenologic diagnosis of fibrous dysplasia should be unequivocal.

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DISCUSSION

Dr. Merrill C. Sosman, Boston, Mass. This interesting paper that Dr. Wyatt has given has been of particular interest to me because we have seen so many cases of this disease localized to the skull, and the problem there in differential diagnosis is often quite difficult. This condition is very apt to involve the ascending plate of the frontal bone as well as the horizontal plate or the supraorbital plate, and sometimes the entire orbit itself on one side. That usually produces a unilateral exophthalmos, often with depression of the eyeball, and it may simulate very closely the diffuse bone involvement of a meningioma. The most important differential point in that diagnosis is that these cases usually show up in children, whereas the meningioma is very rarely found in a young person; they more commonly occur in people thirty or forty years of age or more.

We have gradually learned more and more about this fibrous dysplasia, and as we learn more about it we are able to put many diverse and rare conditions into this same group. "Leontiasis ossea," the irregular overgrowth of skull bones, which Pierre Marie and his group so named fifty or sixty years ago is one. In all probability, the great majority of these belong in the fibrous dysplasia group.

Another one is the ossifying fibroma of the maxillary bone. One sometimes gets an extensive, very opaque overgrowth of bone or osteoid tissue involving one antrum, and this, too, I think, belongs in this same group of fibrous dysplasia.

There is one complication we have seen, particularly in the skull, and that is the development and sometimes a fairly rapid growth of a cystic area in this dense, abnormal bone. If the cyst is biopsied, the chances are about four out of five that one will get a diagnosis of a giant cell tumor, because the cysts usually are lined with giant cells, but they are usually foreign body giant cells and not true giant cell tumors. Interestingly enough, one apparently can stop the growth of these cysts and promote healing by roentgen therapy to the cyst itself.

Dr. Wyatt mentioned Albright's syndrome. I think Albright's syndrome should be limited to the group of cases where there are bone lesions, not necessarily involving more than one bone, but bone lesions of this type associated with sexual precosity and pigmented areas in the skin. That is the syndrome described by Fuller Albright and I think we should limit the name to that syndrome.

Finally, I should like to raise an interesting problem in etiology or pathogenesis which Dr. Thannhauser of the Pratt Clinic in Boston raised several years ago, and that is whether this condition may possibly be a form of neurofibromatosis.

The atypical fibrous tissue could very easily be interpreted by another pathologist as neurogenic tissue, embryonic or undifferentiated nerve tissue. This is associated with pigmented areas in the skin, which as you all know are frequently due to cutaneous neurofibromatosis,

so that I think the suggestion really might be considered seriously.

I would like to ask Dr. Randall if he has any ideas on the possibility that this really might be a neurofibromatosis as Dr. Thannhauser suggested.

Dr. Randall (closing). With regard to whether this is related to generalized neurofibromatosis as brought out by Dr. Thannhauser, the other investigators who have examined a large number of these have found an absence of nerve fibers within the lesions of fibrous dysplasia, and in approximately 9 out of 10 cases of neurofibroma one is able to demonstrate by special stains the presence of nerve fibers or nerve endings. It is largely on this absence of nerve fibers within the lesion of fibrous dysplasia that the support of this theory has not gained widespread acceptance.

REGIONAL ILEITIS (CICATRIZING ENTERITIS) OF PRENATAL ORIGIN

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REVIEW of the literature reveals only one case resembling the one to be presented. The case described previously by Koop, Perlingierio and Weiss,[1] was that of an infant three weeks old. The authors concluded that since the cicatrizing enterocolitis was found in such a young child it seemed likely that the lesion began in prenatal life.

CASE REPORT

Baby K., a white female, was delivered after an uneventful labor on July 9, 1947, and weighed 4 pounds 9 ounces. Almost immediately after birth the child vomited what appeared to be amniotic fluid and vernix caseosa. Vomiting continued intermittently the next day and consisted of large amounts of greenish fluid. The infant was unable to retain anything by mouth. There was now noticeable abdominal distention and the clinical appearance was that of intestinal obstruction. Until this time there had been no bowel movements. There was no evidence of imperforate anus or pyloric stenosis and roentgen studies of the abdomen were ordered.

The findings were those of complete intes-

[1] Koop, C. E., Perlingiero, J.G., and Weiss, W. Cicatrizing enterocolitis in a newborn infant. *Am. J.M. Sc.*, 1947, *214*, 27–33.

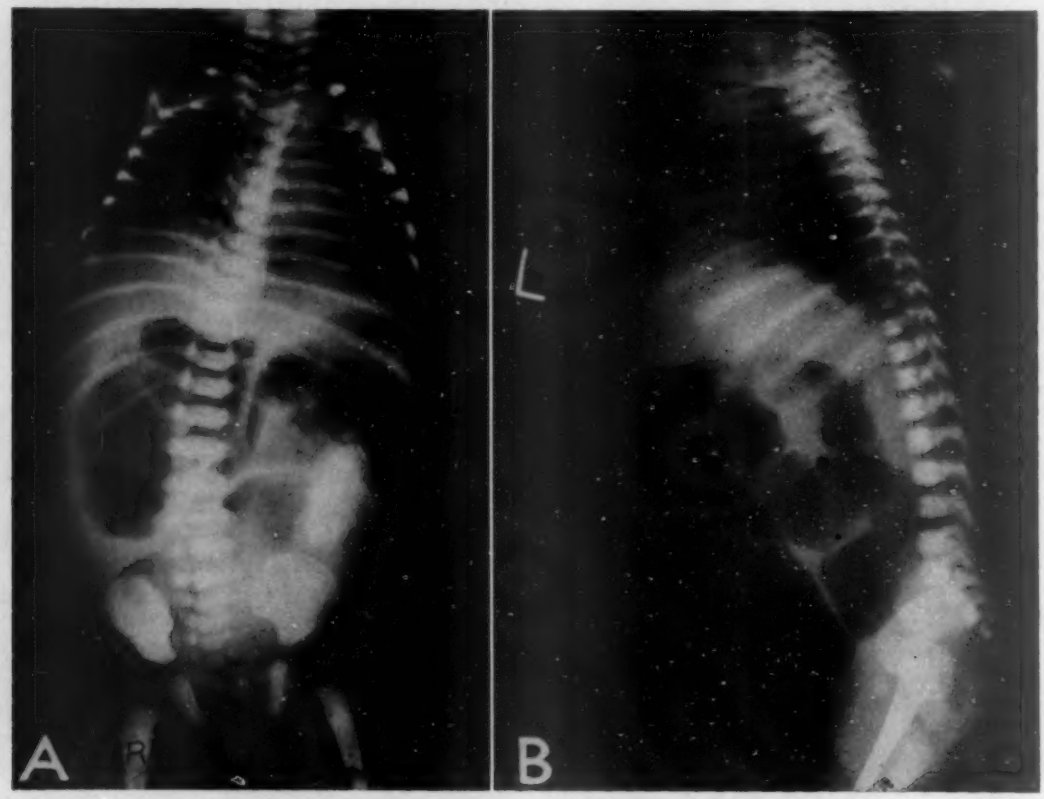

FIG. 1. Examination in dorsal recumbent position (*A*) clearly shows markedly distended loops of small intestine. Large irregularly calcified mass is in left lower abdomen. On lateral view (*B*) the mass is shown to lie anteriorly and higher in abdomen than it was in (*A*).

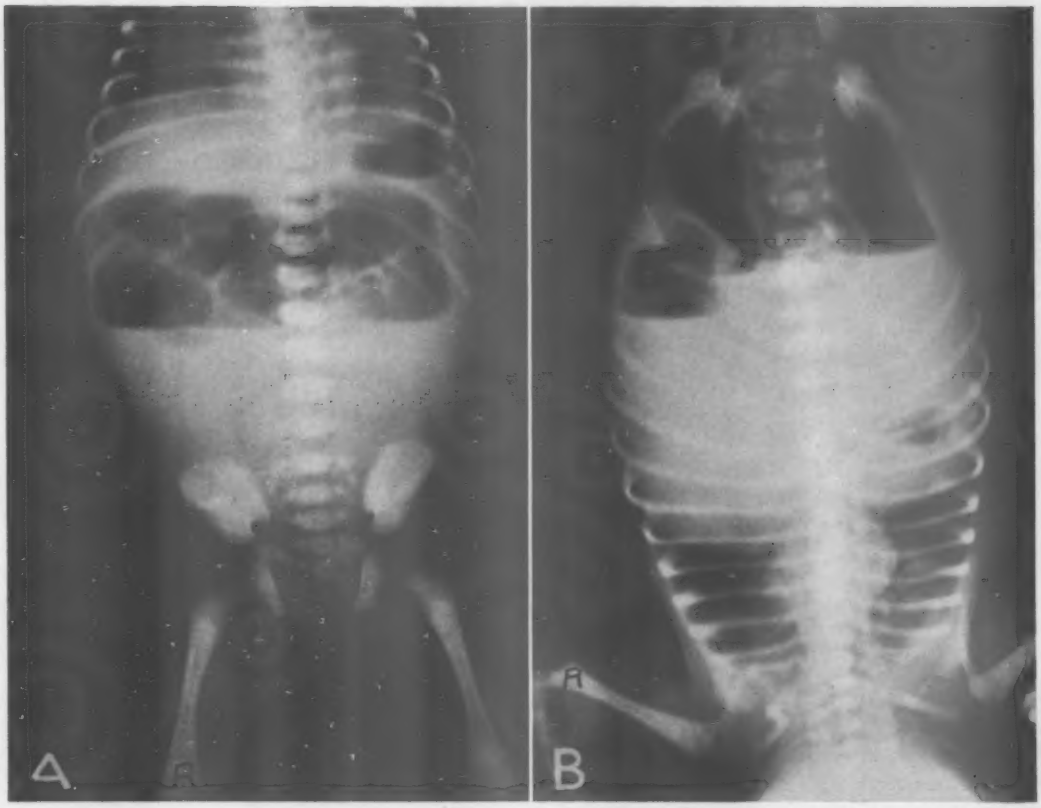

Fig. 2. Erect view (*A*) shows distended small intestine with many fluid levels indicating complete obstruction of small intestine. Portion of calcified mass is visible in gas shadow in left abdomen. Study in inverted position (*B*) reveals similar findings. Calcified area is not as clearly seen in reproduction but could easily be identified on original films.

tinal obstruction. The exact level of obstruction was difficult to ascertain. A most interesting finding was presence of a large, partly calcified, plaque-like mass which seemed to be either in the lumen or wall of distended loops of bowel (Fig. 1 and 2).

The baby continued vomiting and on surgical consultation it was decided to explore the abdomen. On July 11, 1947, an operation was performed.

At operation there was tremendous distention of the small intestine proximal to a lobulated hard yellow mass which measured about 5 by 2.5 by 1 cm. and was located at approximately the mid-portion of the ileum. Distal to this point of obstruction the small and large intestine was collapsed. Just proximal and distal to the mass the ileum was pinched off and was attached to the mass by fibrous adhesions. The mass was resected and the dilated portion of the ileum brought to the surface. This was found to be a blind tip of ileum having been completely occluded by the disease. An ileostomy was done and the abdomen closed. Seven hours postoperatively the baby died.

Autopsy was performed nine hours postmortem.

Autopsy. The body was that of a female infant apparently recently born and although almost full term weighed only 3 pounds 9 ounces. The abdomen showed a left lower quadrant surgical incision from which protruded an open section of small intestine sutured to the abdominal wall and extending not more than 2 cm. from the skin surface. Positive findings were partial atelectasis and congestion of the lungs. The proximal ileum was dilated and showed small areas of hemorrhage. The distal half of the ileum was patent but showed a few areas of narrowing. One area in particular was

almost completely occluded. The cecum and the rest of the large intestine appeared normal.

Gross description of the surgical specimen showed it to consist of an irregular elongated and flattened yellowish-brown piece of tissue. It measured 5 by 2.5 by 1 cm., was firm and had stringy pieces of tissue attached to it. The tissue cut with a gritty resistance and showed yellowish-green material with bands dividing it into compartments. Further section showed that these bands were compressed and narrowed portions of bowel with a hard gritty wall and a soft yellow material in the lumen.

Microscopic examination of the surgical specimen resected from the ileum shows an unusual conglomeration of material consisting of a great deal of calcified substance together with muscular elements suggesting a smooth muscle coat compatible with that seen in the intestinal tract. Parts of the tissue are well vascularized and other areas show an inflammatory process with calcification. There are areas of fibrous tissue proliferation and what appears to be hypertrophy of the muscle. There is also destruction and calcification of the mucosa with calcium in the lumen and throughout the bowel wall.

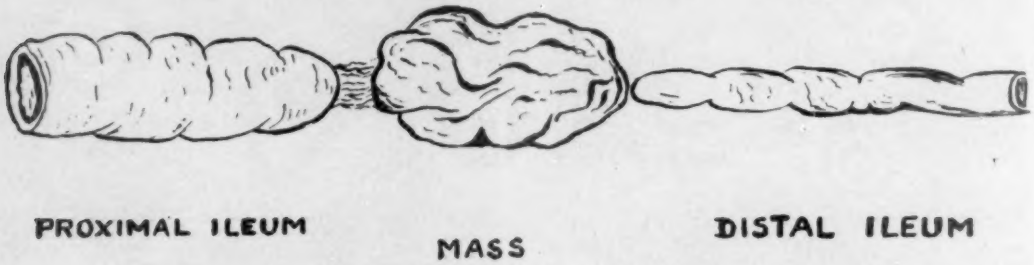

Fig. 3. Schematic diagram of findings at operation. Actually the distention of proximal ileum was much greater than shown on this drawing.

CONCLUSIONS

Since the infant showed a calcified mass in the abdominal cavity when it was three days old and because the ileum showed fibrosis, chronic inflammation and calcium in the bowel wall and lumen it is quite logical to reason that the process began in utero.

SUMMARY

A case which can be classified as regional ileitis or cicatrizing enteritis with calcification has been described. This is the second case to be reported in a newborn and the first case in which the lesion definitely had its origin in utero.

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HODGKIN'S DISEASE

A HISTOPATHOLOGICAL AND CLINICAL CLASSIFICATION WITH RADIOTHERAPEUTIC RESPONSE*

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IT IS the object of this paper to present a classification of Hodgkin's disease, exclusive of Hodgkin's sarcoma, based on a correlation between histopathologic criteria and clinical course, and recognizing three types:

1. Compactly cellular type (slowly progressing)—range of maximum life expectancy forty-eight to 160 months.

2. Fibrogranulomatous type (moderately progressing)—range of maximum life expectancy twenty to sixty months.

3. Loosely cellular type (rapidly progressing)—range of maximum life expectancy twelve to twenty months.

A review of the literature has disclosed no similar classification correlating life expectancy, although several authors[1,3,12,13] have described types of Hodgkin's disease apparently histopathologically similar to those presented here.

The problem of Hodgkin's sarcoma and its relationship to Hodgkin's disease remains unsolved, the former having more of the qualities of a true neoplasm and closely resembling the stem cell lymphoma and clasmatocytic lymphoma of Gall and Mallory[10] from which it can be differentiated only with difficulty by the presence of a few scattered granulomatous foci.

Ever since Hodgkin, in 1832, described the disease which bears his name, its nature has been a debatable one. A brief review of the etiologic literature indicates this uncertainty. Paltauf and Sternberg[15] named the disease lymphogranulomatosis granulomatosa. Benda[15] called it malignant lymphoma and Mallory,[20] Gall and Mallory,[10] and Sugarbaker and Craver[24] included it among the lymphomas. Callender[3] was uncertain whether it is a tumor or reaction to infection, while L'Esperance[17,18] considered it a form of tuberculosis (avian), and Forbus and others related it to brucellosis.[2,9,23] Foot[8] stated that it occupies a position between inflammation and neoplasia and Warren considered the matter controversial[27] although he listed Hodgkin's disease with the lymphomas.[26] Some have favored its virus nature[4] while others have looked upon it as a granulomatous infection, the nature of which is not yet determined.[14,16] Turner and Miller,[21,25] who put Hodgkin's disease, lymphomas, and leukemias in the same category, considered them due to excess or alteration of certain chemical substances in the body, by means of which they report having reproduced these various diseases in animals.

Desjardins[6] noted the frequency with which chronic infection (teeth, tonsils, etc.) is associated with the typical picture of Hodgkin's disease or lymphosarcoma in adjacent nodes. He also included Hodgkin's disease under the term lymphoblastoma, and stated that the relationship between Hodgkin's disease and lymphosarcoma is apparently close.[5]

There has been general agreement for some time on the types of cellular proliferation seen in Hodgkin's disease, but differences of opinion exist on criteria for classification. Gall and Mallory[10] recognized

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Hodgkin's lymphoma and Hodgkin's sarcoma. The former they described as showing polycellular pleomorphism including granulocytes, lymphocytes, plasma cells, eosinophils, clasmatocytes and fibroblasts, along with stem cells and large mononucleated and multinucleated cells having vesicular nuclei and prominent nucleoli. There is a tendency to focal necrosis. They considered Hodgkin's sarcoma as a separate entity, the basic cell of which is the stem cell, or Sternberg-Reed cell. These exhibit marked variability in size and nuclear configuration, and mitotic figures are numerous. Pleomorphism of other small cell types is minimal, and the presence of fibrosis is rare. Foot[7] was in general agreement, calling the two types "granulomatous" and "sarcomatous." Hellwig[11] considered the Gall and Mallory classification of the lymphomas most promising, and believed the average duration of life expectancy of Hodgkin's disease to be about 30 months.

Callender[3] divided the condition into three forms: localized or sclerosing, generalized or cellular, and sarcomatous. His localized type is characterized by gradually increasing fibrosis with typical pleomorphism and Sternberg-Reed cell formation and variable necrosis, while the generalized form shows little sclerosis or necrosis, few typical Sternberg-Reed cells, and abundance of uninuclear reticulum cells. He considered the sarcomatous form very similar to reticulum cell sarcoma.

Jackson's classification[12,13] included early Hodgkin's (paragranuloma), classical Hodgkin's (granuloma), and Hodgkin's sarcoma. The early type was described as superficially resembling lymphocytoma, rather than true Hodgkin's granuloma, with few typical Reed cells and with no fibrosis or eosinophilia. This type is said to run a slow course and may remain unaltered for twenty years, but gradually passes over into the classical form. The latter Jackson described as the typical granulomatous picture, with Sternberg-Reed cells, fibrosis, necrosis and eosinophilic infiltration. Life duration is said to be about 2.6 years. The Hodgkin's sarcoma of Jackson consists mostly of large, round, rather uniform cells with basophilic cytoplasm and prominent nucleoli. Sternberg-Reed cells are present and essential for the diagnosis, but fibrosis, necrosis, granulocytes and eosinophils are absent. Life expectancy is said to be about one year.

Foot[8] described the histopathologic picture as running the gamut from a granulomatous to a neoplastic lesion, and Longcope[19] has stated that there is increasing connective tissue formation as the disease progresses from early to late stage.

Bersack[1] classified Hodgkin's disease into three types histopathologically. His Hodgkin's lymphoreticuloma showed prevalence of reticulum cells, or frequent mitoses, or both; he believed that large or medium sized lymphocytes with mitoses have the same significance. The cases showing the typical features of pleomorphism, Sternberg-Reed cells, and moderate fibrosis he called Hodgkin's granuloma, these making up the main part of his material. His third type, Hodgkin's lymphoma, was described as demonstrating destruction of the gland architecture, with only slight tendency to pleomorphism, and with moderate persistence or prevalence of lymphocytes, mostly of the small variety. He further subdivided the lymphoreticuloma and the lymphoma varieties into typical and atypical types, depending on the presence, or absence, respectively, of Sternberg-Reed cells.

Cohn and Richter[4] and Krumbhaar[16] believed that the average duration of life expectancy is two to three years. The latter stated that fulminating cases may live only a few months, and that more slowly progressive cases may live six to eight years or more, a few on record having a duration of twenty-six years.

In general, the opinions expressed in the various papers quoted above do not directly oppose the presently reported concept of the fundamental changes in Hodgkin's disease. However, according to Murray and Broders,[22] the histopathologic grade of malignancy in Hodgkin's disease bears no relationship to the time of survival follow-

ing histopathologic diagnosis, and the survival rate is the same for all grades. The present study does not bear out their conclusion, but rather suggests that the histopathologic picture and clinical course can be correlated. Each of the 24 cases available for study had microsections, reasonable clinical records, and surgical, medical, or radiotherapeutic data.

It is realized that these 24 cases, in themselves, are insufficient for the drawing of final conclusions. For this reason the method of study employed, described below, was designed to utilize these cases as a test of the impressions of histopathologic classification and prognostic criteria previously gained during a number of years of observation.

Complete clinical abstracts of the cases together with their subsequent histories were made. The microsections were presented, one case at a time, and in no particular order, to the microscopist, who had no previous access to either the case records or the slides. He was given no information concerning the names of patients, previous diagnoses, or clinical data. Solely on histopathologic grounds, therefore, the microscopist made a diagnosis and stated his impression as to clinical course and life expectancy. These were recorded, and later checked against the known clinical facts.

The slides were examined a second time, with the microscopist still having no knowledges of the case history, and still in no definite order, to see whether the original impression could be verified. In those few cases where a discrepancy existed between the first and second readings the slides were presented a third time. The rare persisting discrepancies were then called to the attention of the microscopist, the clinical data of that case made known to him, and the slides critically analyzed in the light of all available information and compared with other cases of similar type, in an effort to explain the discrepancy rationally and to arrive at a final definite diagnosis.

The entire data were charted, all cases which might be expected to have similar course and life expectancy, as judged by cytologic criteria, being grouped together. The expected course was then compared with the known course, and a check on the accuracy of the cytologic criteria was thus established.

Of the 24 cases studied, 19 were followed sufficiently long to adequately determine the clinical course. Of these, 17 confirmed the histopathologic prognostication. A detailed account of the histopathologic criteria and individual case reports follow.

I. Compactly Cellular (Slowly Progressing) Hodgkin's Disease (11 Cases)

(*a*) *Histopathologic Picture* (Fig. 1). The architecture of the lymph node is altered and there are irregular proliferations of reticulo-endothelial cells with formation of Sternberg-Reed cells. Pleomorphism, although present, is not striking. The pro-

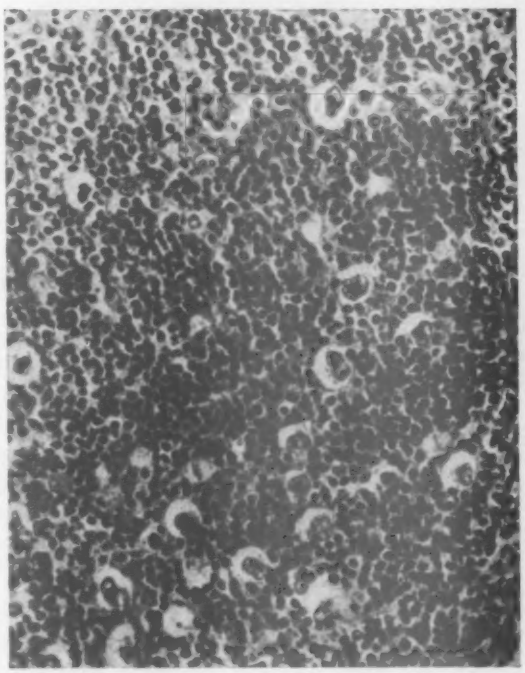

Fig. 1. No. B-1076. Hodgkin's disease—the compactly cellular type (slowly progressing). The compact cellularity is due mostly to lymphoid cells. Eosinophils and plasma cells are present in small numbers. The Sternberg-Reed cells are quite numerous and well formed. The tissue shows little fibrosis. Hematoxylin and eosin stain. ×400.

liferation is mostly of lymphoid and reticulo-endothelial nature with a compact structure. Few eosinophiles and plasma cells are found scattered throughout the section. Fibrosis and necrosis are not marked.

(b) *Cytologic Differential Features.* The compact structure of the tumor with only slight fibrosis are characteristics of this type.

(c) *Correlation between Histopathologic Prognostication and Clinical Course.* Of the 11 cases studied, 7 were followed sufficiently long to determine the course. Of these, 5 followed the histopathologically expected course, while one was more compatible with the acute and one with the fibrogranulomatous type.

(d) *Case Histories.*

Case 1 (Fig. 1). W. S. B. (No. 26877), a white male, aged seventy-two, was admitted to the hospital with a chief complaint of vague abdominal pain and constipation of one month's duration. Essential physical findings were right upper quadrant and left upper quadrant abdominal masses and palpable liver edge. There was no palpable lymphadenopathy at the time of initial examination, with glands appearing in the cervical region one year later. Laboratory findings were essentially normal. A gastrointestinal series suggested findings as seen in carcinoma of the head of the pancreas.

Exploratory laparotomy revealed extensive retroperitoneal tumor, biopsy of which showed Hodgkin's disease of the compactly cellular (slowly progressing) type. Roentgen therapy in divided air doses, totalling 4,800 r to the pelvis, 6,410 r to the abdomen and 675 r to the cervical glands, given over a period of two years, resulted in satisfactory objective and subjective improvement, with good control of the tumor. The patient lived twelve and a half years after onset of symptoms.

Case 2. C. T. (No. 60406), a white male, aged ten, was admitted with a history of drowsiness, fever, malaise, anorexia, headache, and sore throat for one week. He had had a lump in the right axilla for three years which had been gradually enlarging only during the previous six months. Essential physical findings showed a large, firm, irregular, painless, nontender mass in the right axilla, with discrete palpable nodes in cervical, inguinal, and left axillary regions. There was injection of the pharyngeal mucous membrane with red hypertrophied tonsils, and the skin of face, thorax, and scalp showed a reddish maculopapular rash. The tips of liver and spleen were palpable. During the early period of hospitalization the patient's temperature fluctuated between normal and 103° F., with curve similar to the Pel-Ebstein type.

Laboratory examinations showed mild anemia with an initial high white blood cell count which returned to normal after the pharyngitis had subsided. The initial smear showed "11 per cent pathologic lymphocytes." There was a positive heterophile antibody titer (1:40). The urine was essentially negative.

Biopsy of the axillary mass showed Hodgkin's disease of the type classified as compactly cellular (slowly progressing). Roentgen therapy included fractionated dosage (measured in air) over a period of three years and eight months totalling 800 r to the right axilla, 1,550 r to the cervical region and 700 r to various other palpable glands. The response was excellent both subjectively and objectively.

The patient was last seen almost twelve years after onset of symptoms at which time he felt entirely well and exhibited only a few small asymptomatic inguinal nodes.

Case 3. A. B. (No. B-2740), a colored female, aged forty-three, complained of swelling of the neck for five months, and vaginal bleeding and discharge of three weeks' duration, associated with low abdominal pain. Examination showed generalized, firm, moderately tender palpable lymphadenopathy, most marked in the cervical region. No definite information concerning the duration of the adenopathy was available. Fever was minimal with only an occasional rise to 100° F. Laboratory examinations were normal except for a total serum protein of 8.4 grams, with 3.9 grams of albumin and 4.5 grams of globulin, and an erythrocyte sedimentation rate of 26 mm. per hour. Uterine dilatation and curettage showed no malignancy, and vaginal bleeding and discharge were relieved. Biopsy of a cervical gland showed Hodgkin's disease of the compactly cellular type, associated with caseous tuberculous lesions.

The patient received 900 r (measured in air) to each side of the neck in fractionated doses, with disappearance of the nodes. There was recurrence after seven months, and 300 r was given to each side of the neck, after which she

failed to return for further treatment. A follow-up letter twenty-five months after the nodes were first noted revealed that the patient was still alive with nodes present in neck and axilla.

Case 4. W. H., a white male, aged twenty-two, gave a one month history of swelling of the neck. Examination showed left cervical adenopathy, which on biopsy proved to be Hodgkin's disease of the compactly cellular type. Fractionated roentgen therapy, totalling 2,400 r (measured in air) to this area resulted in complete regression of the nodes, with no recurrence at twenty months after onset.

Case 5. R. J., (No. 41921), a colored male, aged ten, was admitted with a one year history of swelling of the right side of the neck. Examination showed generalized lymphadenopathy, most marked in the right cervical region. Biopsy showed compactly cellular type of Hodgkin's disease. The patient received 590 r of fractionated roentgen therapy to the right cervical glands with disappearance of the nodes.

About two and a half years later he was admitted again with left upper quadrant pain, a markedly enlarged hard spleen, and shot-like generalized adenopathy. Splenectomy was done, with histopathologic examination showing Hodgkin's disease as above. He received fractionated doses (measured in air) totalling 1,000 r to the retroperitoneal region, and 200 r each to both inguinal and both cervical areas, with excellent response. At 129 months after onset of symptoms the patient was alive and well with only a few scattered shot-like nodes palpable.

Case 6. R. C., a colored female, aged forty-four, complained of swelling of the neck for one year. Examination showed palpable left cervical nodes which, on biopsy, proved to be Hodgkin's disease of the compactly cellular type. Fractionated dose of 900 r (measured in air) was given resulting in marked improvement. Seven months later she was readmitted with low back pain, malaise, and septic type of fever. There was no palpable adenopathy. Total body irradiation with 300 r resulted in alleviation of symptoms. Nine months later the symptoms recurred with right inguinal adenopathy and an enlarged liver. The retroperitoneal and inguinal regions each received a total of 1,300 r in, air, with relief of symptoms and disappearance of the adenopathy. At twenty-nine months after onset of the disease the patient was living and well.

Case 7. G. O. (No. A-22430), a white male, aged forty-nine, complained of pain in the left upper quadrant and right axilla, and non-productive cough for one month. He had lost 26 pounds over an indefinite period of time. Examination showed a tender mass in the epigastrium and left upper quadrant. There were palpable inguinal and axillary nodes. Roentgenogram of the chest showed an infiltrative lesion in the right lung. Temperature fluctuated between normal and 101° F. daily. One month later the patient developed pain in the low back and right hip. Biopsy of a lymph node showed Hodgkin's disease of the compactly cellular type. Fractionated dose (measured in air) of roentgen therapy totalled 2,200 r to the abdomen, 1,600 r to the right inguinal region, 700 r to the left inguinal region, 800 r to the right hip, and 1,400 r to the lumbar spine. There was no significant response, but rapid retrogressive course and death four and a half months after onset of symptoms.

Case 8. P. B. (No. 39783), a colored female, aged ten, had noted swelling of the cervical glands one month before admission, and cough for one week. On examination the right cervical glands were enlarged to the size of an egg, and were discrete and doughy in consistency. There was no other palpable adenopathy. The temperature rose as high as 103° F. daily. Roentgen examination of the chest showed dense hilar shadows. At various later stages of the disease pulmonary infiltration, an enlarged spleen, and evidence of retroperitoneal involvement were noted.

Biopsy of a lymph node showed Hodgkin's disease of the compactly cellular type. Irradiation totalling 3,790 r to the neck, 1,420 r to the mediastinum, 1,050 r to the abdomen, 1,800 r to the spleen, 850 r to the pelvis, and 1,000 r to miscellaneous lymph nodes was given over a period of six years, with excellent control of the disease until the later stages. The patient died eighty-four and a half months after onset of symptoms.

Case 9. D. E. (No. 9663), a colored female, aged nineteen, gave a six months' history of swelling in the left neck and axilla. Examination revealed large, hard, matted, non-tender left cervical and axillary adenopathy, and smaller discrete nodes in the inguinal and supraclavicular regions. There was poor oral hygiene with dental caries, and a generalized macular pigmented dermatitis. Fever was

only occasional and mild. The white blood cell count was 17,500 with 74 per cent polymorphonuclear leukocytes and 3 per cent eosinophils.

Biopsy of a lymph node showed compactly cellular type of Hodgkin's disease. Fractionated dosage (measured in air) totalling 1,800 r to the left neck and supraclavicular regions and 270 r to the left axilla were given. Good response was obtained, but the patient failed to return for further examination or treatment. A follow-up letter disclosed that the patient was living at forty-seven months after onset of illness, but had become gradually weaker.

Case 10. G. B. (No. A-46950), a colored male, aged six, had had a swelling of the left side of the neck for four and a half months. Examination showed palpable cervical, axillary and inguinal adenopathy, soft, discrete, painless non-tender, and up to 1.5 cm. in diameter. The tonsils were large and nodular but not acutely inflamed. The temperature showed daily rise to 100° F. Laboratory examinations were essentially negative, and roentgen examination of the chest showed nodular densities at both hila. Biopsy of a lymph node revealed compactly cellular type of Hodgkin's disease.

Roentgen therapy totalled 1,600 r to the left neck and 800 r to the right cervical region, and 600 r to the anterior chest. Response was excellent and the patient remained well for twenty-seven months, at which time he was readmitted with Pel-Ebstein type of fever to 104° F., anemia, ascites, splenic enlargement, and marked prominence of hilar nodes. Death occurred shortly thereafter, the total duration of illness having been thirty-five and a quarter months.

Case 11. B. S. (No. 45424), a colored male, aged forty, complained of itching of the skin at night, swelling of cervical and axillary nodes and a sore toe, all of one year's duration. Examination showed generalized non-tender adenopathy and multiple diffuse subcutaneous nodules. There was infection of the right big toe. The tonsils were large and red, and there was pharyngeal injection. Temperature rose intermittently to 102° F. Total serum protein was 6.5 grams with 3.5 grams albumin and 3.0 grams globulin. The urine showed one plus albumin. Roentgen examination of the chest showed healed primary tuberculosis and an infiltrative lesion of the left lower lobe.

Biopsies of the skin revealed mycosis fungoides, and of a node compactly cellular type of Hodgkin's disease. The patient received roentgen therapy totalling 5,000 r to multiple fields during a period of one month, with some improvement in his condition. He was last known to be living ten months after onset of symptoms, but no satisfactory follow up could be obtained.

II. Fibrogranulomatous (moderately progressing) Hodgkin's disease (6 cases)

(a) *Histopathologic Picture* (Fig. 2). The typical picture of Hodgkin's granuloma is seen. There is proliferation of reticuloendothelial cells, with Sternberg-Reed cells, and pleomorphism with abundance of eosinophils, plasma cells and other leukocytes. There is a tendency to fibrosis and necrosis.

(b) *Cytologic Differential Features.* As

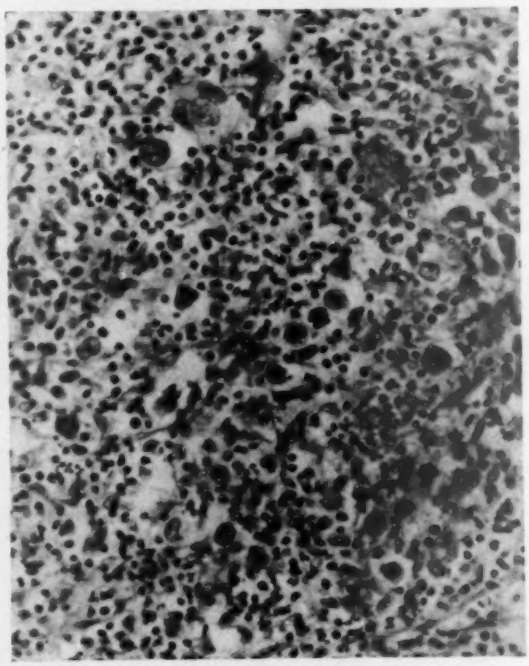

Fig. 2. No. C-1874. Hodgkin's disease—the fibrogranulomatous type (moderately progressing). In this type the cellular pleomorphism is very marked. Eosinophils and plasma cells are numerous. There is active hyperplasia of the reticuloendothelial cells with abundance of Sternberg-Reed cells. Mitosis is frequent. Sclerosing granulation tissue is diffuse and in the larger Hodgkin's areas, foci of necrosis are usually observed. Hematoxylin and eosin stain. ×400.

the name implies, the granulomatous pleomorphism plus the tendency to fibrosis and necrosis differentiates this type.

(c) *Correlation between Histopathologic Prognostication and Clinical Course.* All of the 6 cases were followed adequately, and showed a clinical course in keeping with that expected of a fibrogranulomatous type of Hodgkin's disease.

(d) *Case Histories.*

Case 12 (Fig. 2). O. S. (No. A-8305), a white female, aged thirty-four, complained of a painless, non-tender, firm mass in the right cervical region, and a cough for six months. She had had an eczematoid dermatitis of the hands, forearms, feet and ankles for five years. The only essential findings on physical examination were the cervical mass, associated with palpable cervical and supraclavicular lymph nodes, and the rash as described. At various stages of her later illness she developed axillary and further cervical nodes and roentgenologic evidence of mediastinal and pulmonary involvement. Laboratory data were essentially negative. Lymph node biopsy showed Hodgkin's disease of the fibrogranulomatous type.

The patient has received over 17,000 r (measured in air) to various fields in divided doses during a period of five years. Response of the various recurrent nodes was excellent at first, with gradual decrease in radiosensitivity. At five and a half years after onset of symptoms the patient is in very poor condition, and shows increasing evidence of deterioration.

Case 13. R. J. (No. 61615), a white male, aged forty-nine, gave a thirty months' history of dysphagia, general lymphadenopathy and slight dyspnea. There had been weight loss of 70 pounds during the previous year. Physical examination showed generalized, discrete, firm, non-tender lymphadenopathy, with nodes described as reaching the size of a "marble." There were benign buccal warts on the tongue and palate (histopathologic diagnosis). The tip of the spleen was palpable.

Laboratory examinations were essentially negative except for the white blood cell picture, which showed a count of 25,900, with 70 per cent lymphocytes, 28 per cent polymorphonuclear leukocytes, 1 per cent monocytes, and 1 per cent basophils. There were many smudges and abnormal lymphocytes reported on the smear. Total serum protein was 5.5 gm., with 4.0 gm. albumin and 1.5 gm. globulin. Biopsy of a lymph node showed fibrogranulomatous type of Hodgkin's disease.

The patient had received several previous series of roentgen therapy elsewhere totalling approximately 1,600 r. In this department he received a total of 3,450 r to multiple fields in fractionated dosage over a period of fifteen months. Hemoglobin was reduced to 18 per cent at one time, but improved with transfusions. There was definite improvement during the time the patient was under observation, with multiple small nodes remaining at the time he was last seen. A follow-up inquiry disclosed that death had occurred forty-one months after onset of symptoms.

Case 14. C. W. (No. 48211), a white female, aged forty-four, complained of tinnitus and deafness of the right ear, and pain on the right side of the face and scalp of fifteen months' duration. Examination showed bilateral firm tender submandibular nodes, deafness in the right ear, low grade fever, and suggestive evidence of a right cerebellopontine angle tumor. The only positive laboratory finding was one plus urinary albumin. Roentgen examinations of the skull and chest showed no pathologic changes. Exploratory craniotomy revealed no tumor, and right trigeminal nerve section was done with relief of pain. Biopsy of a lymph node showed Hodgkin's disease of the fibrogranulomatous type. Fractionated radiation therapy totalled 600 r to the enlarged nodes, with some reduction in size of these nodes when the patient was last seen three months later. Inquiry revealed that she had died forty-three months after onset of symptoms.

Case 15. L. H. (No. A-44971), a colored female, aged forty-six, gave a twelve months' history of progressively enlarging painless nodes in the right cervical region, cough, weakness and malaise. She was a known mild diabetic. On examination there were discrete palpable cervical, inguinal and axillary lymph nodes, with a large tender right cervical mass, apparently made up of matted nodes. Uterine fibroids were noted. The temperature curve showed irregular daily spikes as high as 104° F. The red blood cell count was 3,300,000 with 60 per cent hemoglobin and the white blood cell count was 11,020 with 70 per cent polymorphonuclear leukocytes, 17 per cent lymphocytes and 13 per cent monocytes. The lymphocytes were reported to be large.

A lymph node biopsy showed fibrogranulomatous type of Hodgkin's disease. Fractionated roentgen therapy was given, totalling 1,000 r to the anterior and 800 r to the posterior mediastinum, and 400 r to the cervical nodes. There was good immediate response with recurrence after one month, but the patient failed to return for further therapy. Death occurred nineteen months after onset of symptoms.

Case 16. E. S. (No. A-30241), a colored female, aged twenty-eight, noted swelling in the left neck and axilla immediately following postpartum mastitis forty-eight months before admission. During the last six months there had been nausea, anorexia, and weight loss of 40 pounds. Examination revealed palpable, discrete, firm, freely movable cervical, axillary, infraclavicular, inguinal, and pre-auricular nodes, widened mediastinum, palpable liver and spleen, and a poorly demarcated mid-abdominal mass. In the upper outer left breast there were several nodules draining a milky fluid. Temperature rose irregularly as high as 102° F. The red blood cell count was 3,830,000 with 42 per cent hemoglobin and the white blood cell count was 29,800 with 4 per cent lymphocytes, 1 per cent monocytes, and 95 per cent polymorphonuclear leukocytes showing marked shift to the left. Bone marrow study showed myeloid hyperplasia with increased megakaryocytes. The total serum protein was 8.3 gm. with 4.7 gm. albumin and 3.6 gm. globulin. Urinary findings included one plus albumin, one plus glucose, 25–30 leukocytes and 5–10 erythrocytes per high power field. Cultures of the drainage from the breast nodules were sterile, and a brucellergin skin test was positive. Roentgenogram of the chest showed prominent hilar shadows.

Biopsy of a lymph node revealed Hodgkin's disease of the fibrogranulomatous type. Roentgen therapy totalled 1,000 r to the cervical region, 200 r to each axilla, and 200 r to the spleen with excellent response noted at the time this treatment was completed. The patient failed to return for a check-up, but inquiry revealed that she had died sixty-three months after onset of symptoms.

Case 17. N. B. (No. 46673), a colored female, aged thirty-five, gave a four months' history of swelling under the left arm and in the neck, aching of the left arm and frontal headache. Examination showed hard, non-tender, discrete nodes in the left axilla, and left supraclavicular and cervical regions. Temperature ranged intermittently from normal to 103° F. There was a primary luetic lesion of the vagina and serology was positive. Urinary albumin was one plus with 5–8 leukocytes and 1–2 erythrocytes per high power field. Other laboratory findings were within normal limits. Roentgen examination of the chest showed a dense right hilar shadow.

Biopsy of a lymph node revealed fibrogranulomatous type of Hodgkin's disease. Roentgen therapy totalled 1,020 r to the left supraclavicular region, 550 r to the left cervical and left axillary glands, and 250 r to the chest. Response was excellent for one year with recurrence and retrogressive course thereafter. The patient died twenty months after onset of symptoms, and autopsy disclosed widespread Hodgkin's disease and associated pulmonary and lymph node tuberculosis. Cultures of various nodes for *Brucella* were negative.

III. Loosely Cellular (Rapidly Progressing) Hodgkin's Disease (7 Cases)

(a) *Histopathologic Picture* (Fig. 3). The architecture of the node is completely destroyed and replaced by sheets of loose reticulo-endothelial cells with numerous attempts at primitive Sternberg-Reed cell formation evidenced by early nuclear lobulation and with abundant mitotic figures. There is a diffuse pleomorphic picture of eosinophils, plasma cells, lymphocytes and leukocytes of all types with loose edematous structure. The capsule may be invaded and there may also be invasion of lymphatics and blood vessels with an altered blood picture. The tumor is markedly vascular.

(b) *Cytologic Differential Features.* The loose edematous structure, immature type of Sternberg-Reed cells and numerous mitoses are characteristic. Invasion of capsule, vessels and lymphatics also attest to the acute nature of the disease.

(c) *Correlation between Histopathologic Prognostication and Clinical Course.* Of the 7 cases studied, 6 closely followed the expected course. One patient was lost track of before definite conclusions could be drawn.

(d) *Case Histories.*

Case 18 (Fig. 3). S. K. (No. A-41571), an asthmatic and rheumatoid arthritic of long standing, was admitted with a two weeks' history of an enlarging painless lump in the left axilla. Physical findings showed the moderately firm, nodular, fixed, non-tender mass in the left axilla, about the size of a "goose egg." There was mild cardiac enlargement, with asthmatic squeaks in both lungs. The liver was palpable 2 inches, and the spleen 1 inch below the costal margin. During the period of hospitalization there was only occasional rise of temperature to 100° F.

Laboratory examinations were essentially normal except for hemoglobin of 70 per cent, and a rare white blood cell in the urine. Roentgenogram of the chest showed very slight mediastinal widening. Biopsy of a lymph node showed Hodgkin's disease of the loosely cellular (rapidly progressing) type.

The patient received 1,000 r, in air, in divided doses over a period of five days, with no appreciable response and with death six weeks after onset of symptoms.

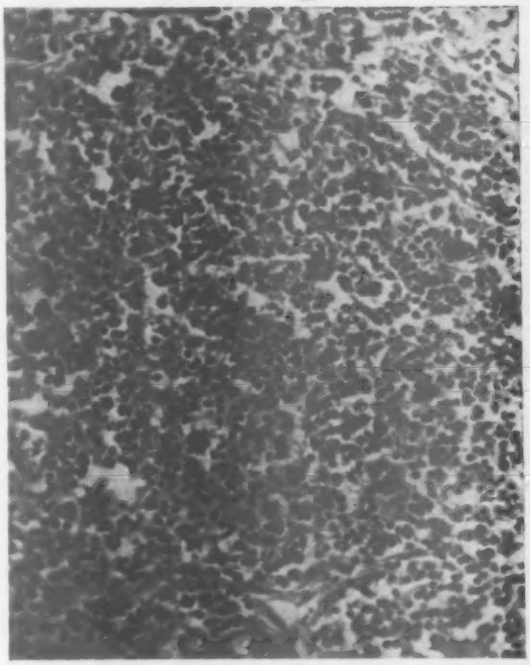

Fig. 3. No. C-9315. Hodgkin's disease—the loosely cellular type (rapidly progressing). This type is characterized by marked cellular pleomorphism. Eosinophils and plasma cells are irregularly distributed. Reticulo-endothelial hyperplasia is very active with numerous mitotic figures. The Sternberg-Reed cells are poorly developed. The stroma is very loose and rich in blood supply. Hematoxylin and eosin stain. ×400.

Case 19. M. S. (No. 42286), a white female, aged thirty-five, gave a nine months' history of painful swelling of the neck and scalp, weight loss, nausea, cough, dyspnea, and facial edema. Essential physical findings were firm, tender cervical, left supraclavicular and right inguinal glands, and tender swelling over the left parietal region. The mediastinum was 16 cm. wide to percussion, and there were bronchial breathing and increased voice sounds at the left intrascapular region. There was irregular spiking of fever to 102° F.

Laboratory examinations showed 60 per cent hemoglobin, with 3,050,000 erythrocytes. The white blood cell count was 8,050, with 82 per cent polymorphonuclear leukocytes (shift to the left), 6 per cent lymphocytes, 8 per cent monocytes, 2 per cent eosinophils, and 2 per cent basophils. Urinary findings included a trace of albumin, one plus acetone, and one to two leukocytes per high power field. Roentgen examination showed a markedly widened mediastinum, and there was erosion of the parietal bone adjacent to the scalp lesion. Biopsy revealed Hodgkin's disease of the loosely cellular type.

Over a period of four months the patient received doses of 1,100 r (measured in air) to the scalp, 2,184 r to the mediastinum, 500 r to the cervical nodes, and 500 r to the right inguinal nodes. There was definite improvement for two months with recurrence and rapid retrogressive course thereafter. Death occurred thirteen months after onset of symptoms.

Case 20. T. A. (No. A-44286), a white male, aged fifty-six, had noted onset of abdominal pain and swelling, and a mass in the left cervical region two months previously. Examination showed discrete left cervical, and right axillary glands, and a nodular liver extending 4 inches beyond the costal margin. There was abdominal distention and evidence of peritoneal fluid. Fever was minimal, with only occasional rise to 101° F. Urinary albumin was one plus, with other laboratory findings essentially normal. A roentgenogram of the chest showed enlarged hilar nodes.

Biopsy revealed Hodgkin's disease of the loosely cellular type. Roentgen therapy totalled 3,200 r to the abdomen, 1,000 r to the neck, and 400 r to the mediastinum, but response was only transient with decrease in size of the nodes,

recurrence, and retrogressive course. Death occurred seven months after onset of symptoms.

Case 21. R. T. (No. A-7928), a white male, aged fifty, complained of swelling of the neck for three months, with moderate associated dyspnea and dysphagia. Examination showed firm, discrete cervical, pre-auricular, axillary and inguinal nodes, 1 to 5 cm. in diameter, most marked in the neck. The tonsils were large. Temperature curve showed an occasional rise to 102° F. Laboratory examinations and roentgenogram of the chest were essentially negative.

Lymph node biopsy showed loosely cellular Hodgkin's disease. Fractionated roentgen therapy to cervical, inguinal, axillary, and supraclavicular glands totalled 5,400 r, with fair response and reduction in size of the nodes. When last seen, six months after the onset of the illness, the patient felt fairly well but there had been an increase in the size and number of palpable nodes.

Case 22. G. N. (No. 30989), a colored male, aged fifty-four, was admitted with a three months' history of fever, pain in the back and abdomen, generalized glandular swelling, 20 pounds weight loss, malaise, pruritus, and headache. Examination showed generalized, discrete, non-tender lymphadenopathy, with the skin of the lower extremities, chest and left shoulder warm, dry and cracked. There were dental caries, hypertrophied tonsils, dullness to percussion and diminished breath sounds in the lower right chest, palpable liver (2 to 3 cm. below the costal margin), and palpable tip of the spleen, with tenderness in the upper abdomen and left flank. Fever varied from normal to as high as 104° F. daily. There was a mild hypochromic anemia. A roentgenogram of the chest showed hilar densities suggesting lymphoma. A diagnosis of loosely cellular Hodgkin's disease was indicated by lymph node biopsy, which was followed by fractionated roentgen therapy totalling 800 r each to chest, left supraclavicular, each cervical, and each inguinal region, and 250 r to each epitrochlear region. There was some clinical improvement noted for about one month, with reduction in size of the nodes, but relapse occurred, with pleural fluid seen on roentgenographic examination. The course was rapidly retrogressive with death four and a half months after onset of symptoms.

Case 23. H. G. (No. 83199), a colored male, aged sixty-four, gave an eleven months' history of pruritus, painful nodules over the entire body, and pain in the back. Examination revealed tender nodes in the cervical, inguinal and axillary regions up to 5 cm. in diameter, multiple pea-sized subcutaneous nodules, heart slightly enlarged to the left, dental caries and poor oral hygiene. The temperature rose irregularly to 102° F. White blood cell count was 14,000 with 21 per cent eosinophils, and the urine contained a trace of albumin.

Biopsy of a lymph node showed loosely cellular type of Hodgkin's disease. Fractionated roentgen therapy totalling 2,300 r was given to multiple fields with satisfactory control of the disease for one year, at which time the patient developed an abdominal mass and palpable liver and spleen. There was gradual deterioration, and death occurred two years after onset of symptoms.

Case 24. W. H., a white male, aged fifty-eight, complained of an "infected throat and tonsils" of seven weeks' duration. Examination showed hypertrophied infected tonsils and generalized lymphadenopathy. The red blood cell count was 3,450,000, with 68 per cent hemoglobin, and white blood cell count was 18,500, with 42 per cent polymorphonuclear leukocytes, 3 per cent lymphocytes, 8 per cent monocytes and 47 per cent plasma cells. Lymph node biopsy showed loosely cellular type of Hodgkin's disease. Fractionated roentgen therapy totalled 1,000 r to the cervical glands, 600 r to the mediastinum, 400 r to each axilla, and 600 r to each inguinal region. There was only transient response, with rapid decline and death five and a quarter months after onset of symptoms.

SUMMARY AND CONCLUSIONS

1. A brief review of the literature relative to opinions concerning etiology and classification of Hodgkin's disease is given.

2. A report is made of the study of twenty-four cases of Hodgkin's disease with the object of showing that careful evaluation of the histopathologic picture is helpful in the prognostication of course and life expectancy.

3. A classification of Hodgkin's disease is offered, based on histopathologic criteria and approximate range of maximum life expectancy:

(a) Compactly cellular type (slowly progressing)—forty-eight to 160 months.

(b) Fibrogranulomatous type (moderately progressing)—twenty to sixty months.

(c) Loosely cellular type (rapidly progressing)—twelve to twenty months.

4. The histopathologic criteria are described.

5. Case histories, with response to roentgen irradiation, and with correlation between the histopathologically expected course and actual course as noted in this series of twenty-four cases, are presented.

6. Nineteen of the twenty-four cases were followed adequately. Of these nineteen cases the courses of seventeen verified the histopathologic prognostication.

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THE ROENTGEN TREATMENT OF CUTANEOUS CARCINOMA INVOLVING CARTILAGE*

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THE perpetuation of false concepts has been commented upon by numerous writers, both medical and lay. Many doctors, including even some radiologists, cling to the opinion that cutaneous cancer which involves cartilage should not be treated by radiotherapeutic methods. Their feeling seems to be that when such a complication has arisen, any attempt to cure the disease by irradiation will inevitably result in perichondritis. This is admittedly a very painful condition and one to be avoided if possible. Some authors urge surgical treatment of all lesions involving cartilage. Others[2,4] advocate the use of heavily filtered protracted irradiation, with relatively high voltage. The results which they have published have been most encouraging. The excellent paper by Driver and Cole[3] emphasizes that "The presence of cartilage in uncomplicated cases does not contraindicate the use of properly selected radiation therapy." In Ackerman and del Regato's recent book[1] the same statement is made.

FIG. 1. Case 1. *A*, basal cell cancer involving cartilage. Treated in January, 1941, by fractionated doses of roentgen rays, 1,500 r being given three times within a period of one week. The total dosage was 4,500 r. *B*, after treatment. The patient had remained well for more than five years and her death was due to unrelated causes.

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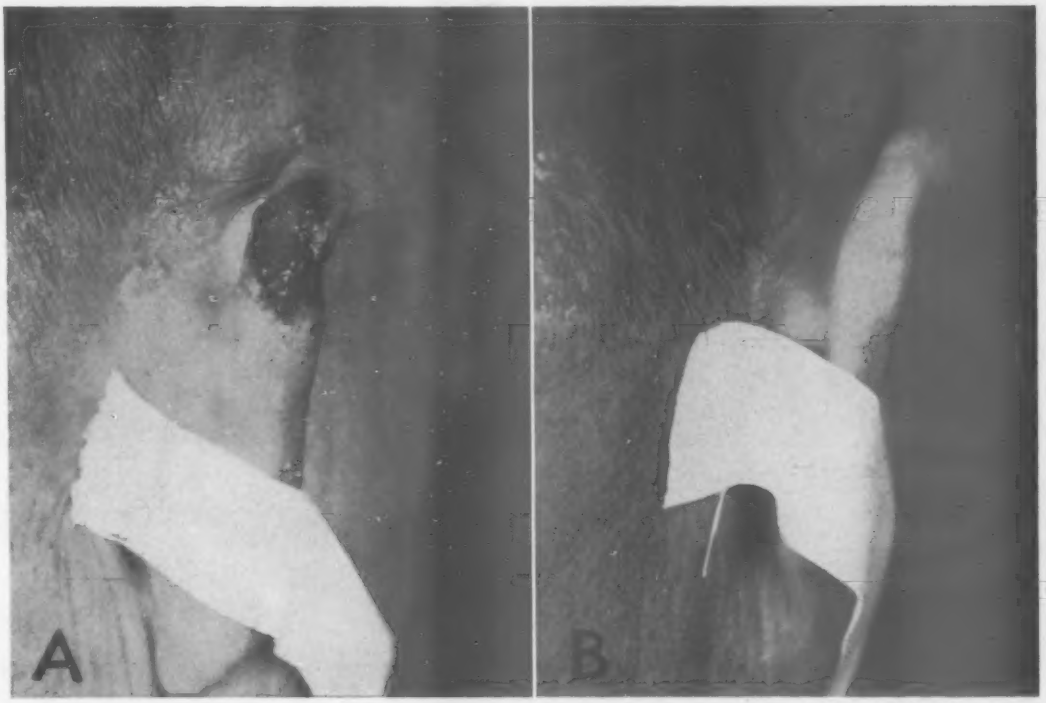

Fig. 2. Case II. *A*, ulcerating basal cell carcinoma involving cartilage of the ear. Treated three times within a week (105 kv.), twice using a filter of 2 mm. Al and for the last treatment 4 mm. Al. The total dosage (air) was 4,500 r. *B*, after treatment. The patient has remained without evidence of disease for over seven years.

Ullmann[5] is in complete agreement with del Regato and writes that he has successfully treated cases involving cartilage or bone.

In the past seven years I have treated several cases in which cutaneous carcinoma has involved cartilage and have employed orthodox methods not unlike those used for the more common types of epitheliomas. The report of some of these cases forms the subject of this communication.

CASE REPORTS

Case I. M.N., a white female, aged sixty-seven, was presented to the Tumor Board of this hospital on October 30, 1940, with a history that for four years she had noted a "sore" on the left ear, which gradually grew in size. Examination revealed a 2 by 1 cm. crusted lesion on the pinna of the left ear which, on biopsy, was reported as "basal cell carcinoma." The patient did not return until January 8, 1941, at which time treatment was started.

Technical factors: voltage, 105 kv. (peak), current 5 ma., target skin distance 30 cm. On January 8, the patient was given 1,500 r through 1 mm. Al; on January 10 she received another 1,500 r, using 2 mm. Al as filter; and on January 14, 1941, her final treatment of 1,500 r was given, employing 4 mm. Al as filter. All measurements are in air.

The cutaneous reaction to these treatments was a mild though moist but not very painful epithelitis. Vaseline was advised and the ear healed within a month. The patient remained without evidence of aural disease for more than five years when she died of cancer of the cervix.

Case II. L.J., a white male, aged fifty-six, once a medical student (unsuccessful) but more recently a vendor of patent medicines, came to the Tumor Board of this hospital in February, 1940. He complained of an ulcer of the right ear lobe. This ulcer had been increasing in size for two years and, on examination, measured 3 by 1.5 cm. Biopsy of the lesion was reported as "basal cell carcinoma." The original recommendation of the Board was for removal of that portion of the ear which appeared involved.

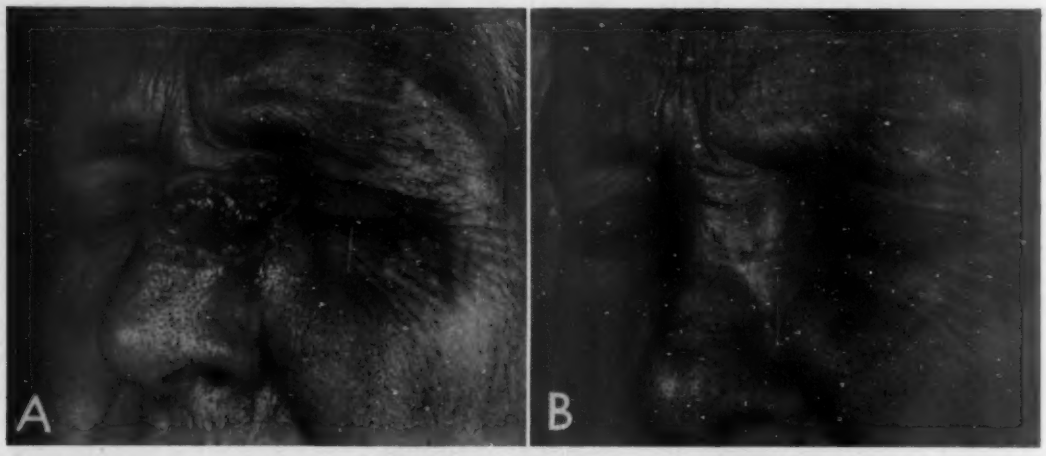

Fig. 3. Case III. *A*, squamous cell carcinoma invading the cartilage of the nose and inner canthus, treated in September, 1943. A daily dose of 200 r was employed. *B*, after treatment. This patient has remained free of evidence of disease for almost five years.

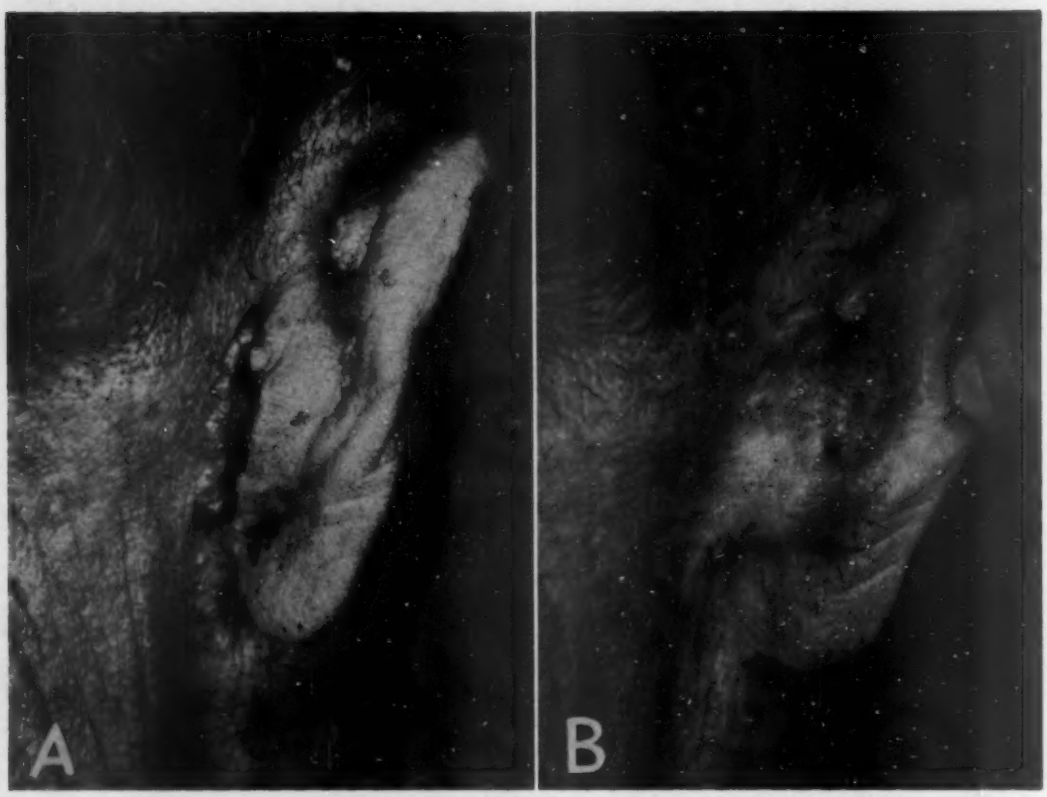

Fig. 4. Case IV. *A*, deeply ulcerating basal cell carcinoma with cartilage involvement. Treated daily for a total of 5,000 r (air). *B*, after treatment. The patient has remained well over six years.

This the patient declined, maintaining that as a "public speaker" he wished to avoid disfigurement and he was referred instead for radiation therapy.

Treatment: Patient received 1,500 r (air) on March 3, and like amounts on March 6 and March 8, 1940. The factors were: voltage, 105 kv. (peak), 5 ma.; target skin distance 30 cm. The first two treatments were given through a filter of 2 mm. Al and for the final one 4 mm. Al was employed. The reaction to this was a brisk, dry erythema which healed within six weeks. At no time did the patient complain of pain. There has been no evidence of disease for more than seven years.

In retrospect, though the result was quite satisfactory both from a cosmetic standpoint and as far as eliminating the carcinoma was concerned, we would now be inclined to use considerably lighter filtration throughout the treatments.

FIG. 5. Case V. *A*, bleeding, ulcerated adenocarcinoma involving cartilage of the ear. Treated three times within a week. The total dosage (air) was 4,500 r. *B*, after treatment. The patient remained without evidence of disease until his death from unrelated causes two years later.

CASE III. C. B., a white seamstress, aged fifty-four, came to the Tumor Board September 22, 1943, with a history that for nine years she had noted a gradually enlarging ulcer on the left side of her nose. The lesion measured approximately 4 by 3.5 cm. in diameter. It was completely fixed to the underlying bone and also to the inner canthus of the left eye. However, no bone pathology was demonstrable roentgenographically. Biopsy from this lesion was reported as "squamous cell carcinoma, Grade 3." There was no regional lymphadenopathy and the patient was referred for radiation therapy.

Treatment: A special portal was cut from 1.5 mm. lead and through this the patient received 200 r (air) daily with the following factors: 200 kv., 20 ma., target skin distance 40 cm., filter 1.0 Cu and 2 mm. Al, half-value layer 1.0 mm. Cu. This was continued for thirty-four treatment days, with a total of 5,000 r on October 28, 1943. The cutaneous response to these treatments was a moderate dry erythema and remarkable improvement in the appearance of the tumor. The skin promptly healed and she has remained well over five years.

CASE IV. F. G., a white male, aged sixty-one, came to the hospital Tumor Board in September, 1941, with a history that for more than three years his family had noted a slowly growing ulceration behind his right ear which, on

Fig. 6. Case vi. *A* and *B*, extensive basal cell carcinoma of the nose, showing fungating and destructive lesion. Treated every other day in increments of 1,500 r (air) for three treatments. *C* and *D*, after treatment. The patient continues to remain well after more than eight years.

examination, measured approximately 4 by 0.5 cm. The patient did not wear glasses. A biopsy revealed "basal cell carcinoma" and he was referred for radiation therapy despite the obvious cartilage involvement.

Treatment: The factors were: 200 kv., 20 ma., target skin distance 50 cm., filter 1.0 Cu and 2 mm. Al with half-value layer 1.0 mm. Cu. A special portal was cut and 200 r (air) were given daily for five days each week to a total of 5,000 r. Treatment was concluded October 17, 1941, and a brisk though dry and painless erythema resulted which subsided within the next two weeks. The patient has remained well for more than six years.

Case V. H. G., a white male, aged eighty, was referred to the Tumor Board of this hospital in June, 1940, from the Dermatology Service. In the concha of the left ear there could be seen a bleeding, ulcerated tumor, 3 cm. in diameter, with a clean base. The patient gave a

history that for three years he had received all sorts of nostrums to this ear, including the use of "cancer paste." He was somewhat vague about the exact nature of the "treatments" he had received but was clear that each of them had caused him considerable pain. The deformity of his ear was a silent confirmatory witness to this statement. Biopsy showed "adenocarcinoma, Grade 3, perhaps of sweat gland origin." There was no regional adenopathy. He was referred for radiation therapy.

Treatment: A special portal was cut in lead 1.5 mm. thick and within one week the patient received the following: 1,500 r (air) on June 18 and like amounts on June 20 and June 22, 1940. The factors were: voltage, 105 kv. (peak), 5 ma., target skin distance 30 cm. The first two treatments were given through a filter of 2 mm. Al and for the final treatment 4 mm. Al was employed. Only a moderate dry erythema developed which promptly subsided and was asymptomatic. The patient remained without evidence of disease until his death from unrelated causes two years later.

Case vi. S. S., a white female, aged eighty-two, gave the hospital Tumor Board, in September, 1939, a history of having had a "pimple" on her nose seven years previously. This would ulcerate and a serous discharge would escape from it with subsequent crust formation. This series of events was repeated every few days. In August, 1938, the rate of growth became accelerated and the patient consulted a physician for the first time. He made a clinical diagnosis of "skin cancer" and so informed the patient. She did nothing about this, however, for more than a year. At that time she yielded to the pleas of her friends and family to have something done about her growing deformity, and she came to the hospital for treatment. The ulceration at that time measured 4 cm. in diameter. Biopsy of the lesion showed "basal cell carcinoma" and she was referred by the Tumor Board of this hospital for radiation therapy. She has continued to remain well for more than seven years.

Treatment: Patient received 1,500 r (air) on September 25, and like amounts on September 27 and September 29, 1939. The factors were: voltage, 105 kv. (peak), 5 ma., target skin distance 30 cm. The first of these three treatments was given through a filter of 1 mm. Al and the other two were given through a filter of 4 mm. Al. The reaction to this was a brisk moist erythema and the nose appeared completely healed on her clinic visit on June 12, 1941. She complained but little of pain at any time. In June, 1946, she developed an independent lesion, also basal cell carcinoma, on her forehead. The latter also responded well to treatment.

DISCUSSION

The treatment of epithelioma which involves cartilage requires individualization, but it is not a radiologic *noli me tangere*. Excellent results have been obtained not only with surgery but also with a wide variety of irradiation techniques. High voltage radiation using heavy filtration, radium and low voltage treatments have all met with success, depending on the site and type of the lesion and the competence of the therapist.

Many patients with cancer of the skin may be successfully treated by roentgen irradiation though cartilage or even bone is involved in the cancerous process. We have found useful both the methods of high voltage roentgen rays[2,4] and especially that described by Widmann,[6] employing low voltage roentgen rays.

We cannot be arbitrary about the alleged superiority of any one method of treatment. For a large number of lesions involving cartilage either high or low voltage irradiation appears satisfactory. The exclusively surgical treatment of these tumors does not appear justified. Proper irradiation in many such cases yields a substantial number of gratifying results.

SUMMARY

Six typical cases of cutaneous carcinoma involving cartilage have been presented. In four of these, the treatment was similar to that employed by us in the majority of skin cancers.

In two cases high voltage (200 kv.) and fairly heavy filtration (1.0 mm. Cu) were used. The results in all have been satisfactory but no single technique has unquestionable advantage over other treatment plans.

None of these patients developed peri-

chondritis and it seems as though the fear of this complication has been exaggerated. It certainly is not inevitable. Should it occur, surgery can then be employed.

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Vol. 61, No. 3

THE TREATMENT OF RADIATION SICKNESS WITH ADRENAL CORTICAL HORMONE (DESOXYCORTICOSTERONE ACETATE)*

A PRELIMINARY REPORT ON FIFTY CASES

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AMONG the many therapeutic agents employed in the treatment of radiation sickness, the use of desoxycorticosterone acetate (DCA), the synthetic hormone of the adrenal cortex, appears well founded, not only on theoretical grounds but also on the basis of the results of recent animal experimentation. Most of the symptoms of radiation sickness can be explained on the basis of intoxication of the irradiated body by tissue decomposition products, histamine-like in character, if not histamine itself.[1] It is known that desoxycorticosterone acetate counteracts certain toxic effects of histamine.[2] An association between liver function and radiation sickness has been inferred from the fact that the latter is more pronounced after irradiation of the upper abdomen. In recent work, one of the authors (F.E.) was able to demonstrate that desoxycorticosterone acetate protects the liver against irradiation-induced fatty changes.[3] This protective action was accompanied by a decrease in the mortality rate produced in mice with the roentgen-ray doses employed in these studies.[4] These investigations offered a rational basis for the clinical use of desoxycorticosterone acetate in the treatment of radiation sickness.

MATERIALS AND METHODS

A preliminary account of our clinical experience with the use of desoxycorticosterone acetate in the therapy of radiation sickness is presented in this paper. A total of 50 patients, all males but one, comprise the clinical material of this study (Table I). The ages of the patients varied from twenty to sixty-five (Table II). These patients were irradiated for a variety of benign and malignant conditions. The sections of the body irradiated are listed in Table III. All of the patients exhibited gastrointestinal manifestations, in addition to other symptoms of radiation sickness. The first symptoms appeared at variable intervals, from one to thirty-three days following the beginning of irradiation. However, 36 out of the 50 patients exhibited the initial symptoms within the first four days. The amount of radiation producing these symptoms of radiation varied from a single dose of 50 r (measured in air), (Case 27) to a total cumulative dose of 7,500 r (measured in air) (Case 8). The correlation of daily and total cumulative dose with the onset, frequency and severity of symptoms will be presented at a later date with a larger series of cases.

Precise clinical studies of radiation sick-

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TABLE I

Case No.	Name	Age	Diagnosis	Section Treated	Radiation Therapy Date Started	Radiation Sickness Date Onset	Cortate Therapy				Remarks
							Date		Symptoms		
							Started	Ended	Before DCA	After DCA	
1	A.R.	30	Marie-Strümpell arthritis	Trunk	2/17/47	2/18/47	2/19/47	2/21/47	Headaches x Dizziness x Weakness x Lost appetite x Nausea x Diarrhea x	Lost appetite x	
2	A.J.	48	Carcinoma of nares	Head and Neck	4/ 1/47	4/ 7/47	4/11/47	4/14/47	Dizziness x Weakness x Weakness x Lost appetite xxx Nausea xx	No symptoms	Two recurrences of radiation sickness—vomiting relieved by DCA
3	A.J.	26	Carcinoma of testis	Trunk	2/17/47	2/20/47	2/21/47	2/28/47	Headaches x Lost appetite x Vomiting x	Headaches	In a 3 month period—5 recurrences of radiation sickness—vomiting, each time, relieved by DCA
4	B.J.	46	Lymphosarcoma, generalized	Trunk	2/ 2/47	2/ 6/47	2/14/47	2/17/47	Lost appetite x Nausea x	Lost appetite	One recurrence of radiation sickness—vomiting relieved by DCA
5	B.M.	36	Osteochondrosarcoma	Abdomen; extremities	3/25/47	3/28/47	4/ 9/47	4/13/47	Headaches x Weakness x Diarrhea x	Headaches x	
6	B.G.	29	Lymphosarcoma, generalized	Abdomen	12/ 7/47	12/ 9/46	12/ 9/46	12/12/46	Lost appetite x Nausea x	No symptoms	
7	B.F.	53	Bronchogenic carcinoma, generalized	Thorax	2/12/47	2/15/47	2/17/47	2/18/47	Dizziness Nausea xx	Nausea x	One recurrence of radiation sickness. Nausea relieved by DCA again
8	C.F.	52	Lymphosarcoma, generalized	Abdomen	1/24/47	1/27/47	2/12/47	2/15/47	Headaches x Weakness x Lost appetite x Nausea x Vomiting x	No symptoms	Three recurrences of radiation sickness—nausea, vomiting relieved by DCA again
9	C.Th.	29	Lymphopathia venereum with lymphadenopathy	Abdomen Groin	4/24/47	4/25/47	4/30/47	5/ 2/47	Headaches x Dizziness x Weakness x Lost appetite x Nausea x Vomiting xx	Headaches x Weakness x Lost appetite ?	Massive involvement of deep inguinal and pelvic nodes by lymphopathia venereum infection

10	Di.L.V.	26	Hodgkin's, generalized	Trunk	2/25/47	2/26/47	2/26/47	2/27/47	Headaches x Lost appetite x Nausea x	No symptoms	
11	D.H.	43	Pulmonary carcinoma	Thorax	12/23/46	12/27/46	1/ 6/47	1/10/47	Dizziness xxx Weakness xxx Lost appetite xxx Nausea xxx Vomiting xxx	Nausea x	
12	F.F.	49	Rectosigmoid carcinoma	Abdomen	2/19/47	2/21/47	2/25/47	2/28/47	Weakness x Lost appetite x Nausea x	No symptoms	One recurrence of radiation sickness. Headache, lost appetite and nausea relieved by DCA again
13	G.H.	44	Breast carcinoma; skeletal metastases	Trunk	11/26/46	12/16/46	1/10/47	1/14/47	Headache xx Lost appetite xx Nausea x Vomiting x	Lost appetite ?	Widespread disease. Downhill course. Heavy morphine analgesia
14	G.E.	36	Antral carcinoma with metastatic neck nodes	Head and Neck	12/30/46	1/ 6/47	1/ 6/47	1/11/47	Lost appetite x Nausea x	Lost appetite x	
15	G.H.	36	Rectal carcinoma	Abdomen	2/26/47	2/28/47	3/ 1/47	3/ 3/47	Lost appetite x Nausea x	No symptoms	
16	M.Ch.	37	Astrocytoma	Head	3/26/47	4/11/47	4/24/47	4/26/47	Headaches xx Lost appetite x Nausea x	Lost appetite x Nausea xx Vomiting x	Downhill clinical course from expanding, inoperable brain tumor
17	H.R.	20	Reticulum cell sarcoma, generalized	Trunk	11/15/46	12/ 6/46	12/ 7/46	12/ 6/46	Weakness xxx Lost appetite xx Vomiting xx Fear xxx	No improvement	Massive involvement. Death from perforated malignant duodenal ulceration
18	K.J.	57	Bronchogenic carcinoma	Thorax	2/26/47	2/28/47	3/ 3/47	3/ 5/47	Weakness x Lost appetite ? Nausea xx	Weakness x Nausea x	
19	L.E.	38	Myeloid leukemia	Abdomen	12/21/46	12/24/46	12/28/46	12/31/46	Lost appetite x Nausea x	No symptoms	
20	L.Wm.	58	Hypernephroma	Abdomen	3/10/47	3/15/47	3/25/47	3/26/47	Headaches x Lost appetite x Nausea x Vomiting x	No symptoms	
21	M.J.	48	Hodgkin's, generalized	Head	3/ 1/47	3/ 1/47	3/ 5/47	3/ 7/47	Headaches x Dizziness x Weakness x Lost appetite x Nausea x Vomiting x	Lost appetite Nausea ?	Abdominal and ileocolic Hodgkin's disease, advanced, progressive
22	M.L.	52	Hyperthyroidism	Neck	2/25/47	3/10/47	3/19/47	3/21/47	Weakness x Lost appetite xx Nausea xx Vomiting xx	Lost appetite xx	

TABLE I—(*continued*)

Case No.	Name	Age	Diagnosis	Section Treated	Radiation Therapy Date Started	Radiation Sickness Date Onset	Cortate Therapy				Remarks
							Date		Symptoms		
							Started	Ended	Before DCA	After DCA	
23	M.J.W.	23	Recitulum cell sarcoma	Thorax	11/30/46	12/ 9/46	12/10/46	12/13/46	Lost appetite x Nausea x	No symptoms	
24	M.G.	25	Hodgkin's disease	Neck	2/24/47	2/27/47	3/24/47	3/26/47	Weakness x Lost appetite xx Nausea x Vomiting x	No symptoms	
25	O'T.H.	54	Lymphosarcoma	Groin	12/27/46	12/31/46	12/31/46	1/ 2/47	Weakness x Lost appetite xx Nausea x Diarrhea x	No symptoms	
26	P.B.	36	Marie-Strümpell arthritis	Trunk	2/ 6/47	2/ 8/47	2/ 8/47	2/11/47	Headache x Lost appetite x Nausea x	No symptoms	
27	P.D.	50	Myelogenous leukemia	Abdomen	2/10/47	2/19/47	2/19/47	2/25/47	Lost appetite x Nausea x Vomiting x Diarrhea	No symptoms	
28	R.B.	25	Rheumatoid arthritis	Trunk	12/ 9/46	12/10/46	12/17/46	12/18/46	Dizziness x Weakness x Vomiting x	Dizziness x	
29	S.A.	49	Cerebellar hemangioblastoma	Head	1/24/47	1/25/47	1/25/47	1/28/47	Weakness x Lost appetite x Vomiting x	Symptoms slightly improved	
30	S.Ch.	31	Nasopharyngeal lympho-epithelioma, neck metastases advanced	Neck	11/ 4/46	11/26/46	12/27/46	12/30/46	Dizziness x Nausea x Vomiting x	Nausea x	One recurrence of radiation sickness—nausea and vomiting slightly relieved by DCA. Downhill course and death
31	S.H.	55	Bronchogenic carcinoma	Trunk	12/19/46	12/23/46	12/27/46	12/28/46	Headaches x Weakness x Lost appetite x Nausea x Insomnia x	No symptoms	
32	S.J.	37	Mediastinal lymphoma	Thorax	1/10/47	1/17/47	1/17/47	1/22/47	Weakness xx Lost appetite x Nausea x	Lost appetite x	Esophageal obstruction by tumor

33	R.A.	32	Hemangioblastoma, cerebellum	Head	6/4/47	6/ 5/47	6/ 7/47	6/10/47	Headaches x Dizziness x Weakness x Lost appetite xx Nausea xx Vomiting xxx	Headaches Dizziness	
34	S.M.	25	Seminoma, testis	Trunk	3/11/47	3/18/47	3/27/47	3/28/47	Nausea xx Vomiting xx	No symptoms	One recurrence of radiation sickness—nausea and vomiting relieved by DCA again
35	TH.R.	65	Lymphatic leukemia	Groin	4/23/47	4/25/47	4/25/47	5/ 1/47	Lost appetite xx Nausea x Vomiting x	Lost appetite x Nausea x	Pulmonary tuberculosis, advanced
36	T.R.	52	Carcinoma kidney, advanced; metastases	Abdomen	4/28/47	4/28/47	4/30/47	5/ 3/47	Weakness x Lost apeptite xxx Nausea xxx Vomiting xxx	Weakness x Lost appetite xx	One recurrence of radiation sickness—nausea and vomiting relieved by DCA again
37	V.W.J.	39	Breast carcinoma (male)	Thorax	1/ 8/47	1/10/47	1/10/47	1/16/47	Lost appetite xx Nausea xx Vomiting x	No symptoms	
38	W.R.	63	Bronchogenic carcinoma; abdominal metastasis	Abdomen	12/10/46	12/11/46	12/16/46	12/19/46	Dizziness x Weakness xx Lost appetite xx Nausea xxx	No symptoms	
39	W.F.	22	Teratoma of the testis	Trunk	3/29/47	4/10/47	4/10/47	4/11/47	Lost appetite x Nausea x	No symptoms	One recurrence of radiation sickness—vomiting relieve by DCA
40	C.G.	21	Embryonal carcinoma of testis	Abdomen	6/ 3/47	6/ 3/47	6/10/47	6/13/47	Lost appetite x Nausea x Vomiting xxx	Lost appetite x Nausea x	
41	O.F.	56	Carcinoma of tonsil	Neck	6/ 5/47	6/ 7/47	6/16/47	6/18/47	Headache xxx Dizziness xx Weakness xx Lost appetite xxx Nausea x Vomiting x	Headache xx Dizziness x Weakness x	
42	B.M.	54	Mediastinal tumor	Thorax	6/25/47	6/28/47	7/ 3/47	7/ 5/47	Dizziness x Weakness x Lost appetite xx Vomiting xx	Headache Dizziness	
43	F.R.	30	Chromophobe adenoma—pituitary	Head	7/15/47	7/16/47	8/ 7/47	8/11/47	Nausea x Vomiting x Lost appetite xx	Dizziness x Lost appetite xx Nausea x	

TABLE I—(*continued*)

Case No.	Name	Age	Diagnosis	Section Treated	Radiation Therapy Date Started	Radiation Sickness Date Onset	Cortate Therapy				Remarks
							Date		Symptoms		
							Started	Ended	Before DCA	After DCA	
44	L.A.	51	Mediastinal tumor, probably lymphoma	Thorax	8/ 4/47	8/ 4/47	8/11/47	8/15/47	Dizziness x Lost appetite xxx Nausea xxx	Dizziness xx Lost appetite xx	
45	P.J.	49	Bronchogenic carcinoma	Thorax	6/27/47	7/ 2/47	7/ 3/47	7/ 7/47	Headache x Dizziness x Weakness x Lost appetite x Nausea x Vomiting x	Symptom free	Downhill clinic course. Two additional courses less effective
46	S.N.	20	Marie-Strümpell arthritis	Trunk	8/ 8/47	8/11/47	8/12/47	8/15/47	Headache x Dizziness x Weakness x Nausea x Vomiting x	Headache x Dizziness x Weakness x Nausea x	
47	S.G.	51	Lymphosarcoma	Abdomen	7/21/47	7/22/47	7/31/47	8/ 3/47	Headache x Dizziness x Weakness x Lost appetite xx Nausea xxx Vomiting xxx	Headache x Dizziness xx Weakness xx Lost appetite xxx Nausea xxx Vomiting xxx	Benadryl and pyridoxine were also ineffective. Clinical evidence of liver involvement by lymphosarcoma
48	W.F.	30	Bronchogenic carcinoma	Thorax	6/18/47	6/21/47	6/21/47	6/24/47	Headache x Lost appetite x Nausea x Vomiting x	Headache x Lost appetite x	One recurrence of radiation sickness—DCA less effective
49	L.B.	49	Myelogenous leukemia	Abdomen	7/25/47	7/25/47	7/28/47	8/ 1/47	Headaches x Dizziness xx Weakness xxx Lost appetite xxx Nausea xxx Insomnia xx	Headaches x Dizziness xxx Weakness xx Insomnia xx	Unusually rapid response to DCA—possible psychic factor in this case
50	L.J.	21	Testicular tumor; abdominal metastases	Abdomen	8/ 8/47	8/ 9/47	8/11/47	8/15/47	Dizziness x Weakness xx Lost appetite xxx Nausea xx Vomiting x	Lost appetite xx	

ness present marked difficulties for a number of reasons. (1) This complication of radiation therapy is a symptom complex involving several systems of the body. (2) Not all the symptoms are experienced by each patient. (3) A patient may develop radiation sickness during one course of irradiation and not during another. (4) The frequency and severity of symptoms varies with the section of the body treated and the amount of daily and total cumulative radiation. These facts make an appraisal of remedies for prophylactic therapy extremely difficult. Therefore, the effectiveness is evaluated only on a therapeutic basis.

In order to ascertain the symptoms of radiation sickness as objectively and accurately as possible, we have prepared a special chart for this problem (Table IV). This table lists the symptoms arranged according to the following five main groups; (1) general; (2) gastrointestinal; (3) blood; (4) cardiovascular; (5) psychic. Each patient's symptoms were daily recorded from the beginning of radiation therapy. The intensity of his symptoms were graded

TABLE II

AGE DISTRIBUTION

Age Group	No.	Case No. in Table I
16–25	9	17, 23, 24, 28, 34, 39, 40, 46, 50
25–36	9	1, 3, 6, 9, 10, 30, 33, 43, 48
36–45	10	5, 11, 13, 14, 15, 16, 19, 26, 32, 37
46–55	17	2, 4, 7, 8, 12, 21, 22, 25, 27, 29, 31, 36, 42, 44, 45, 47, 49
56–65	5	18, 20, 35, 38, 41

as follows: o—no symptoms, ?—questionable, x—positive, xx—pronounced, xxx—very pronounced. Table V summarizes our observations. In this preliminary report we are considering only the symptoms of group 1—general, and group 2—gastrointestinal. Special attention was devoted to the most distressing of all the symptoms, nausea and/or vomiting.

Each patient received in a twenty-four hour period, three doses of 5 mg. each of desoxycorticosterone acetate* in peanut oil at eight hour intervals, intramuscularly. The drug was given until relief of symptoms was obtained, but not for more than five

TABLE III

REVIEW OF BODY SECTIONS TREATED

Section	No.	Case No. in Table I
I Head and neck	11	2, 14, 16, 21, 22, 24, 29, 30, 33, 41, 43
II Thorax	10	7, 11, 18, 23, 32, 37, 42, 44, 45, 48
III Abdomen	15	5, 6, 8, 9, 12, 15, 19, 20, 27, 36, 38, 40, 47, 49, 50
IV Trunk	12	1, 3, 4, 10, 13, 17, 26, 28, 31, 34, 39, 46
V Others	2	25, 35
TOTAL	50	

days, in order to avoid the symptoms of overdosage. In observing these rules no untoward reactions were encountered in any of our patients. In instances where radiation sickness recurred while irradiation was continued, further courses of desoxycorticosterone acetate were given at intervals of three to five days during this period.

RESULTS

Our results obtained in the treatment of radiation sickness with desoxycorticosterone acetate are summarized in Table V. This table reveals that only 3 patients (16, 17 and 47) out of 50 failed to respond to this hormone. Two of the 3 failures were patients suffering from inoperable advanced brain tumors. Of the remaining 47 patients, 37 were completely relieved of the most distressing symptoms, nausea and/or

* Desoxycorticosterone acetate was furnished by the Schering Corp., Bloomfield, New Jersey, through the courtesy of Dr. E. Henderson.

TABLE IV

RADIATION SICKNESS CHART

NAME AGE

DIAGNOSIS WEIGHT

HEIGHT

Date																	
I GENERAL SYMPTOMS a. Headaches																	
b. Dizziness																	
c. Weakness																	
II GASTROINTESTINAL a. Lost appetite																	
b. Nausea																	
c. Vomiting																	
d. Diarrhea																	
III BLOOD a. Hemoglobin																	
b. Leukopenia																	
c. Thrombopenia																	
d. Sedimentation																	
IV CARDIOVASCULAR a. Blood pressure																	
b. Arrhythmia																	
c. Pulse																	
V PSYCHIC SYMPTOMS a. Insomnia																	
b. Fear																	

MEDICATION

GENERAL CONDITION AT START OF TREATMENT

SECTIONS TREATED

GRADES o . . . No symptoms ? . . . Questionable x . . . Positive xx . . . Pronounced xxx . . . Very pronounced

TABLE V

RESULTS OF DCA TREATMENT

Group	Total	Case No. of Table I
I Free of all symptoms	19	2, 6, 8, 10, 12, 15, 19, 20, 23, 24, 25, 26, 27, 31, 34, 37, 38, 39, 45
II Nausea and/or vomiting completely relieved	18	1, 3, 4, 5*, 9, 13, 14, 22, 28, 32, 33, 36, 41, 42, 44, 48, 49, 50
III Nausea and/or vomiting improved	10	7, 11, 18, 21, 29, 30, 35, 40, 43, 46
IV No response	3	16, 17, 47
TOTAL	50	(*) Relieved from diarrhea

vomiting. Nineteen cases were relieved of all symptoms including nausea and/or vomiting. It appears noteworthy that in 12 cases, where nausea and/or vomiting recurred during the course of irradiation, repeated administration of desoxycorticosterone acetate was effective in relieving these symptoms (Table 1, Cases 2, 3, 4, 7, 8, 12, 30, 34, 36, 39, 45, 48). However, if the course of the disease for which the patient was irradiated showed a retrogressive trend, then repeated administration of desoxycorticosterone acetate was less effective (Table 1, Cases 30, 45, 48).

A breakdown of our results with the treatment by desoxycorticosterone acetate according to sections of the body irradiated is given in Table VI. This table shows that the hormone proved most effective where the body section irradiated included the liver. It may be noted that out of 27 cases irradiated over the abdomen and trunk, 23 were completely relieved from nausea and/or vomiting by the use of the hormone.

TABLE VI

CORRELATION OF DCA RESULTS WITH BODY SECTION TREATED

Section	Total Cases Irradiated	Nausea and/or Vomiting Entirely Relieved	Case No.
I Head and neck	11	6	2, 14, 22, 24, 33, 41
II Thorax	10	7	23, 32, 37, 42, 44, 45, 48
III Abdomen	15	13	5, 6, 8, 9, 12, 15, 19, 20, 27, 36, 38, 49, 50
IV Trunk	12	10	1, 3, 4, 10, 13, 26, 28, 41, 34, 39
V Others	2	1	25
TOTAL	50	37	

DISCUSSION

The clinical data presented in this paper demonstrate the clinical efficacy of desoxycorticosterone acetate in the treatment of radiation sickness, although based on a relatively small number of patients. They confirm the conclusions drawn by one of the authors (F.E.) from his animal experiments. In particular, the pronounced effect of desoxycorticosterone acetate in cases where the field of irradiation includes the liver appears noteworthy. This seems in agreement with the animal experimental observation that desoxycorticosterone acetate prevents the roentgen-ray induced fatty changes in this organ. Thus our clinical observations tend to support the opinion that desoxycorticosterone acetate therapy strikes at the etiological factor producing radiation sickness. They are also in agreement with the results obtained by Weichert[5] from the study of 25 exclusively gynecological cases. These studies will be continued and expanded to include evaluation of the effects of desoxycorticosterone acetate on radiation leukopenia and other symptoms.

SUMMARY

The value of desoxycorticosterone acetate (DCA) in the treatment of radiation sickness was studied in a series of 50 patients. All of these cases exhibited nausea and/or vomiting besides other symptoms of radiation sickness. Radiation was administered for a variety of conditions, benign and malignant. Of the 50 patients, only 3 failed to respond to desoxycorticosterone acetate therapy. Thirty-seven patients were completely relieved of nausea

and/or vomiting. These data indicate the effectiveness of desoxycorticosterone acetate in the treatment of radiation sickness.

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DEPTH DOSE MEASUREMENTS FOR 250 KV. ROENTGEN RAYS*

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INTRODUCTION

DURING recent years, there has been a marked increase in the employment of 250 kv. roentgen rays for the treatment of deep-seated lesions. Previous depth dose measurements by Exner and Packard[1] include 250 kv. radiation. However, their equipment differed considerably from the apparatus now used clinically for 250 kv., and their results are presented in a form primarily useful for comparative purposes. It was decided, therefore, to extend our earlier depth dose measurements to include this voltage range.

It has been shown by Mayneord and Lamerton[2] that within normal limits any combination of filter and voltage which results in the same half-value layer also gives nearly identical depth dose values. Therefore, present 200 kv. depth dose tables may be used also for 250 kv. for half-value layers up to 2 mm. Cu. However, most 250 kv. treatments are given with a higher roentgen-ray quality, and our present tables are based, therefore, on measurements with a half-layer value of approximately 3 mm. Cu. This roentgen-ray quality may be obtained either with pulsating potential as used in this study, or with constant potential. In the latter cases the required filtration will obviously be less.

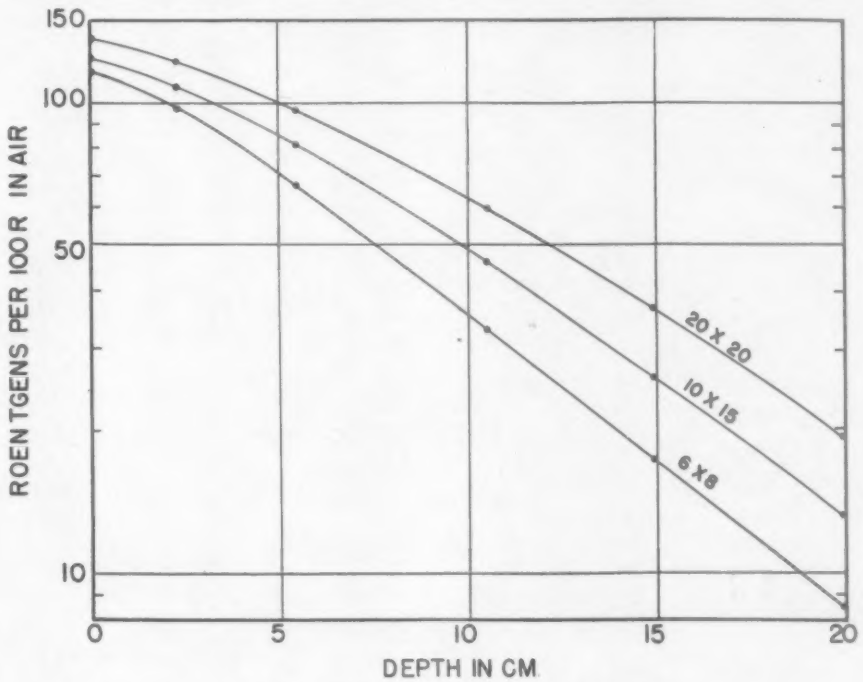

FIG. 1. Results of depth dose measurements.

EXPERIMENTAL SET-UP

The present depth dose measurements were made in a "presdwood" phantom with a density of 0.985 gm. per cc. as pre-

* From the Physics Laboratory, Department of Hospitals, City of New York.

viously described.[3] The dimensions of the phantom were 30 by 30 by 30 cm. The phantom was used also in our 200 kv. depth dose measurements where the results were found to coincide with those we made in a water phantom of the same dimensions. These measurements, furthermore, checked with the extrapolation chamber depth dose

TABLE I

DEPTH DOSE FOR 250 KV. HALF-VALUE LAYER 3.2 MM. CU
(in roentgens per 100 r on skin)

Area (sq. cm.)	5 cm. Diam. 19.6	6×8 48	8×10 80	10×10 100	10×12 120	10×15 150	15×20 300	20×20 400
Target Skin Distance = 50 cm.								
"Air" dose	92	85	83	82	81	81	77	74
Skin dose	100	100	100	100	100	100	100	100
1 cm.	91	94	95	96	96	96	97	98
2	81	86	88	89	90	90	92	93
3	71	77	80	82	83	84	85	87
4	61	68	72	74	75	76	79	81
5	52	60	65	66	68	69	72	75
6	45	52	56	59	60	62	66	68
7	39	46	50	52	53	55	60	63
8	32	40	45	47	48	50	55	57
9	28	35	40	41	42	44	49	52
10	24	31	35	37	38	39	44	47
11	20	26	31	33	33	35	39	42
12	17	23	27	29	29	31	35	38
13	15	21	24	25	26	28	32	33
14	12	17	21	22	23	24	28	30
15	10	15	18	20	20	21	25	27
20	5	8	9	10	11	11	13	15
Target Skin Distance = 70 cm.								
"Air" dose	92	85	83	82	81	81	77	74
Skin dose	100	100	100	100	100	100	100	100
1 cm.	92	95	96	97	97	97	98	99
2	83	87	89	91	91	92	93	94
3	73	80	83	84	85	86	88	90
4	64	71	75	77	78	80	83	85
5	55	62	68	70	71	72	76	79
6	48	56	60	62	63	66	69	73
7	41	50	54	56	58	59	64	67
8	36	44	48	51	52	54	59	62
9	30	38	43	45	46	48	54	56
10	27	34	38	41	42	43	49	51
11	22	29	34	36	37	39	44	47
12	18	26	31	32	33	35	40	42
13	17	23	27	29	30	31	35	39
14	14	20	24	25	26	28	32	34
15	12	18	21	23	24	24	28	30
20	6	9	11	11	13	14	15	18

Table I—*Continued*

Area (sq. cm.)	5 cm. Diam. 19.6	6×8 48	8×10 80	10×10 100	10×12 120	10×15 150	15×20 300	20×20 400
				Target Skin Distance = 80 cm.				
"Air" dose	92	85	83	82	81	81	77	74
Skin dose	100	100	100	100	100	100	100	100
1 cm.	92	95	96	97	97	97	98	99
2	84	88	90	92	92	93	95	96
3	73	80	84	85	86	87	88	90
4	65	73	76	78	80	81	85	87
5	56	64	69	71	72	74	78	80
6	49	56	60	64	65	67	71	73
7	42	50	55	57	59	61	66	70
8	36	45	50	52	53	55	61	63
9	31	39	45	46	47	49	55	58
10	28	35	40	42	44	45	50	53
11	23	31	36	38	39	40	45	49
12	19	27	31	34	35	36	41	44
13	17	24	28	30	32	33	37	40
14	14	21	25	26	27	29	33	36
15	12	19	22	24	24	25	29	32
20	6	9	12	12	13	15	16	19

data by Quimby.[4] The radiation was measured by means of a thimble type ionization chamber having an inside diameter of 0.7 cm., and a volume of approximately 0.8 cc. The ionization chamber was permanently connected to a projection type electrometer which was effectively shielded with lead. Preliminary tests made with the chamber disconnected from the electrometer indicated that the leakage was negligible. The high voltage generator was a commercially available 250 kv. self-rectified, self-contained unit. The inherent filtration of the roentgen tube, oil and plastic window was approximately 2 mm. Al. The half-value layer of 3.2 mm. Cu was obtained by adding a filter of 3.0 mm. Cu plus 1 mm. Al. This filtration corresponds to a Thoraeus filter of approximately 0.6 mm. Sn plus 0.25 mm. Cu plus 1 mm. Al. The beam was collimated by means of a variable diaphragm system located at a target distance of 36.5 cm. Comparative readings made with cones showed no significant difference.

Preliminary tests were made also at 200 kv. to check our earlier depth dose measurements which were made with a different type of equipment. These showed that identical depth dose values were obtained with the present and former experimental set-ups.

RESULTS

The results of our depth dose measurements are shown graphically in Figure 1, and Tables 1 and 11 have been calculated from these data. The figures for the various target skin distances were determined by use of the inverse square law, while those for the other field sizes were obtained by interpolation.

In Table 1, the data are presented in terms of the skin dose to permit direct calculation of the tumor dose in percentage of the radiation received by the skin. The data in Table 11 are given in terms of 100

roentgens "in air" for the radiologists who give their treatments in terms of the air dose.

DISCUSSION

As might be expected, the backscatter factors are considerably reduced by increasing the voltage from 200 to 250 kv. These, as well as our depth dose data, are in general agreement with those of Exner and Packard,[1] though our depth dose values are somewhat higher. This, however, is con-

TABLE II

DEPTH DOSE FOR 250 KV. HALF-VALUE LAYER 3.2 MM. CU
(in roentgens per 100 r "in air")

Area (sq. cm.)	5 cm. Diam. 19.6	6×8 48	8×10 80	10×10 100	10×12 120	10×15 150	15×20 300	20×20 400
Target Skin Distance = 50 cm.								
"Air" dose	100	100	100	100	100	100	100	100
Skin dose	109	117	121	122	123	123	130	135
1 cm.	99	110	115	117	118	118	126	132
2	88	100	106	109	110	111	119	125
3	77	90	97	100	102	103	111	117
4	67	80	87	90	92	94	104	110
5	57	70	78	81	83	85	94	101
6	49	61	68	72	74	76	85	92
7	42	54	61	64	66	68	78	85
8	35	47	54	57	59	61	71	77
9	30	41	48	50	52	54	64	70
10	26	36	42	45	47	48	57	63
11	22	31	37	40	41	43	51	57
12	18	27	33	35	36	38	46	51
13	16	24	29	31	33	34	41	46
14	13	20	25	27	28	30	36	40
15	11	18	22	24	25	26	32	36
20	5	9	11	12	13	14	17	20
Target Skin Distance = 70 cm.								
"Air" dose	100	100	100	100	100	100	100	100
Skin dose	109	117	121	122	123	123	130	135
1 cm.	100	111	116	118	119	119	127	133
2	90	102	108	111	112	113	121	127
3	79	93	100	103	105	106	114	121
4	70	83	90	94	96	98	108	114
5	60	73	82	85	87	89	99	106
6	52	65	72	76	78	81	90	98
7	45	58	65	68	71	73	83	91
8	38	51	58	62	64	66	77	83
9	33	45	52	55	57	59	70	76
10	29	40	46	50	52	53	63	69
11	24	34	41	44	46	48	57	63
12	20	30	37	39	40	43	52	57
13	18	27	33	35	37	38	46	52
14	15	23	29	31	32	34	41	46
15	13	21	25	28	29	30	37	41
20	6	11	13	14	16	17	20	24

TABLE II—*Continued*

Area (sq. cm.)	5 cm. Diam.	6×8	8×10	10×10	10×12	10×15	15×20	20×20
	19.6	48	80	100	120	150	300	400
	Target Skin Distance = 80 cm.							
"Air" dose	100	100	100	100	100	100	100	100
Skin dose	109	117	121	122	123	123	130	135
1 cm.	100	111	116	118	119	119	127	133
2	91	103	109	112	113	114	123	129
3	80	94	101	104	106	107	115	122
4	71	85	92	95	98	100	110	117
5	61	75	83	87	89	91	101	108
6	53	66	73	78	80	82	92	99
7	46	59	67	70	73	75	86	94
8	39	52	60	63	65	68	79	85
9	34	46	54	56	58	60	72	78
10	30	41	48	51	54	55	65	72
11	25	36	43	46	47	49	59	66
12	21	31	38	41	42	44	53	59
13	19	28	34	37	39	40	48	54
14	15	24	30	32	33	36	43	48
15	13	22	26	29	30	31	38	43
20	6	11	14	15	16	18	21	25

sistent with a similar comparison at 200 kv.

The results indicate clearly a definite improvement in the depth dose as the quality is increased from a half-value layer of 2 to 3.2 mm. Cu. For instance, at a depth of 10 cm., the gain is 13 per cent for a 10 by 10 cm. field and 10 per cent for a 20 by 20 cm. field. In other words, it is possible to increase the tumor dose at this depth by these factors for the same exposure to the skin.

630 West 168th St.
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SPOT SCANOGRAPHY

METHOD OF DETERMINING BONE MEASUREMENT

By WILBUR K. MUELLER, M.D.,* *and* JOHN M. HIGGASON, M.D.†
ST. LOUIS, MISSOURI

IT IS frequently requested of the roentgenologist to determine the exact measurement of certain bones. The requests are most often limited to the bones of the lower extremities. It is important to the orthopedic surgeon to know the exact length of these bones when an epiphyseal arrest is contemplated. This is an operative procedure used in any case in which there has been disturbance of the normal epiphyseal growth resulting in a shortening of the affected extremity. Such a shortening is most often seen in cases following poliomyelitis; however, not infrequently it is due to an early fusion of an epiphysis from local infection or trauma. In adults, where epiphyseal growth has ceased, the shortened extremity may be lengthened by certain operative procedures.

The first method to be used for determining exact bone measurements was introduced by Millwee[1] in 1937, and was called "slit scanography." This apparatus consists of a regular roentgenographic table with a side rail and arm to hold a roentgen tube at a film-target distance of 25 inches. This tube is driven lengthwise of the table at varying speeds by a motor driven gear. Instead of the regular circular cone, an adjustable slit is arranged between the tube and patient. Hence, as the tube is passed from one end of the table to the other, a beam of roentgen rays like a narrow sheet or line of roentgen rays traverses the patient. The apparatus is so arranged that all the film is protected by lead except the narrow strip which is being exposed. The principle of this method is that all the central beams of roentgen rays pass through the body and reach the plate from the same angle, hence reducing the effects of distortion and variation of density.

We used a modification of Millwee's method until the introduction of our "spot scanography," which is much simpler, less time consuming and requires no special apparatus. To determine the exact bone measurements of a lower extremity the patient is placed on an ordinary roentgenographic table in supine position with a 14 by 17 inch film under the thigh. Both the hip and knee joint must be included on the film; hence the film will have to be placed obliquely in many cases. Then the roentgen tube with a small circular cone, just large enough to include the hip or knee joint, is centered directly over the head of the femur at a 36 inch distance and an exposure made; then without moving the patient or the film the tube is centered over the distal end of the femur and another exposure made. Another 14 by 17 inch film is placed under the leg, including the knee and ankle joint on the plate and again two small spot views are made, one centering over the proximal end of the tibia and the other centering over the distal end of the tibia.

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† Resident in Radiology, St. Mary's Group of Hospitals.

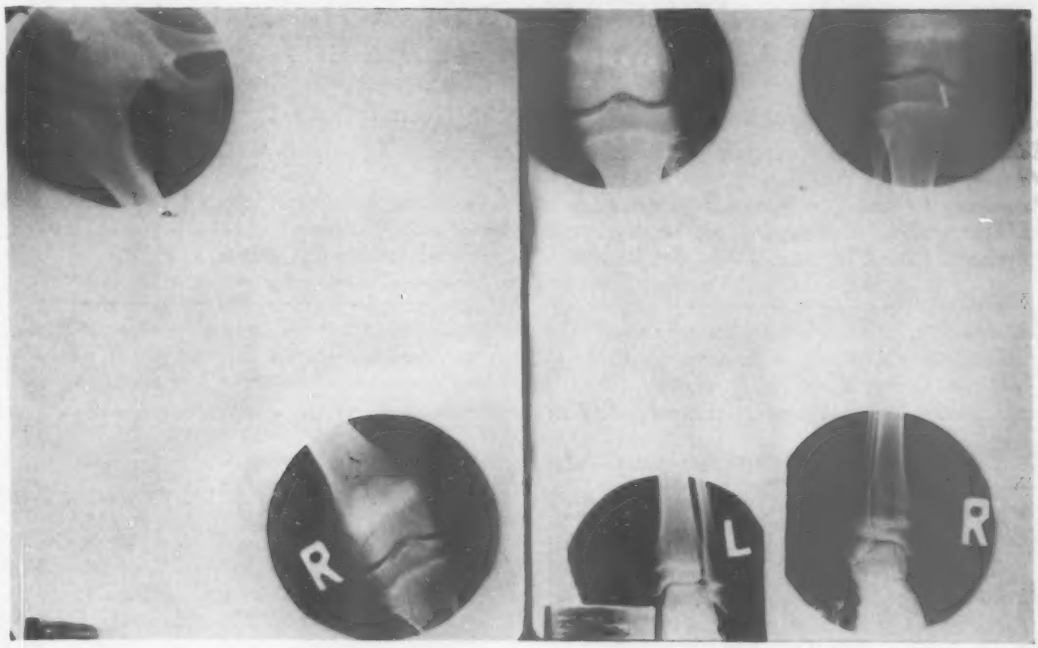

Fig. 1

The roentgenogram will appear as in Figure 1. From these films the measurements are made according to Gill and Abbott[2] as follows: femur, a straight line from the tip of the femoral head to the medial lip of the internal condyle at the joint line; tibia, a straight line from the medial lip of the internal condyle at the joint line to the tip of the internal malleolus.

Bone measurements determined by this method have been compared with those by the slit scanography method and are equally accurate.

This method in our hands has proved to be very simple, just as accurate as other methods and requires no special apparatus.

This method can also be used for determining pelvic measurements.

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THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

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EDITORIALS

THE CLINICAL AND ROENTGENOLOGICAL IMPLICATIONS OF HYALURONIDASE

DURAN-REYNALS,[1] in 1929, while studying the effect of extracts of various organs from normal and immunized animals on the virulence of vaccine virus, unexpectedly discovered that extracts of normal testis increased the infectivity to an extraordinary degree. He also found that the action occurred on the cells of the host rather than on the virus. Later, based on the results of additional investigations, he advanced the theory that the tissue extracts contain a "spreading factor" which is responsible for the enhancement of the infectivity.

It is now generally accepted that this spreading factor is a mucolytic enzyme which in 1936 was identified by Meyer, Dubos and Smith[2] as hyaluronidase. When injected together with the vaccine virus, or a suspension of bacteria or certain foreign material, as for example india ink, this substance increases the permeability of the host tissues. Normally, there is a physiologic barrier which inhibits the spreading of any injected medium. The barrier consists of a hyaluronic acid gel, a viscous mucopolysaccharide of wide distribution. Its presence has been demonstrated in the ground substance of the connective tissue, the vitreous body, umbilical cord, ovarian follicular fluid, synovial fluid, the capsules of certain bacteria, several types of tumors and in the stroma of a number of mammalian organs. The enzyme hydrolyzes the hyaluronic acid gel and by reducing the high viscosity breaks down the physiologic barrier of invasiveness. It has been shown by Duran-Reynals that with optimal hyaluronidase concentration the spreading action is so rapid that the bleb which results from injection of a fluid into the tissues is completely diffused within a minute or less.

Hyaluronidase occurs, apart from testis which in the mammal forms its main source, in bee venom, the venom of poisonous spiders and snakes, leech extract, many bacterial strains and some malignant tumors.

As one may surmise, the "spreading" property of the hyaluronidase incited investigations covering a rather wide range of applicability. In the beginning the chief interest centered on the effect of the enzyme on various infections and the relationship which exists between virulence and hyluronidase formation. It was found that the human serum, normally, contains a factor which partly inactivates the effect of the hyaluronidase. Under specific conditions, as demonstrated by Haas,[3] the body may be stimulated to produce other factors which are either part of the defense mechanism or serve to promote the invasion of bacteria and venoms. However, the problem of preparing suitable antisera to conbat the infections is still in the experimental stage and no definite correlation is as yet possible.

Later the investigations were extended to a study of the role of hyaluronidase in the invasiveness of cancer. Coman[4] who

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had already spent considerable time in identifying other factors of the invasiveness, as for example the decreased adhesiveness and the ameboid movement of cancer cells, was particularly interested in this problem. He performed two kinds of experiments. The first was concerned with the determination of the amounts of hyaluronidase present in the various tumors. It was found that several of the malignant neoplasms examined did contain the enzyme. In some instances the amounts were significant, lending support to the assumption that the spreading factor may facilitate the invasiveness of cancer cells. In a second set of experiments hyaluronidase was injected into the animals to determine whether an excess of enzyme would increase the capacity for invasion. Transplantable sarcomas of mice and virus-induced papillomas of rabbits were used. In neither instance was augmented invasiveness demonstrable. Coman thought that since the injections of the in vivo experiments were made daily over long periods, the formation of antienzymes in the sense described by Haas,[3] may perhaps have accounted for the negative results. He concluded that it is quite possible that hyaluronidase is not requisite for invasive growth, but that when it does operate, it augments the facility with which invasion occurs. Other investigators, as for example Simpson and Gopal-Ayengar,[5] reported that hyaluronidase markedly increased the invasiveness of transplantable carcinomas in mice, while Hakanson[6] observed a definite nonspecific inhibition of hyaluronidase activity by the blood serum in human cancer.

During the past few years hyaluronidase has been used to advantage in the medical field, especially where a quick absorption of subcutaneously injected fluids, drugs and various foreign substances, or a removal of viscuous fluids from the body is desired. Hechter, Dopkeen and Yudell[7] employed the enzyme to facilitate clinical hypodermoclysis. While no direct quantitative tests were possible the results showed that in the cases in which sixty to ninety minutes were required for the swellings produced by the clysis to disappear after cessation of injection, this time was reduced to only ten to twenty minutes by the subcutaneous administration of the enzyme prior to clysis. Kirby, Eckenhoff and Looby[8] studied the effect of hyaluronidase in spreading the action of local anesthetic agents used in nerve block and infiltration anesthesia. They found that the enzyme increased the zone of anesthesia but in view of the accelerated absorption decreased its duration. If epinephrine was added the duration of the anesthesia was restored to that without hyaluronidase and because of the delay in absorption the spreading effect was extended over a larger area. Ragan and De Lamater[9] described the successful injection of hyaluronidase into pathologic joints to reduce the viscosity of the exudate. The same procedure was applied by Meyer[10] to remove thick viscous fluid from the pleural and peritoneal cavities in a patient with mesothelioma.

Of considerable interest is the implication of hyaluronidase in the process of fertilization. Bergenstal and Scott[11] reviewed all the material available on this subject. It has been shown that the ovum in the fallopian tubes is surrounded by many follicular cells which are held together by a viscous gel-like material. The testicular hyaluronidase hydrolyzes this material thereby dispersing the follicular cells and making it possible for the sperm

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to penetrate the denuded ovum. Bergenstal and Scott determined the proportionality between hyaluronidase activity and sperm count and found a definite relationship between the two. The absence of sperm was always associated with absence of the enzyme while all specimens with sperm contained the enzyme. Other investigators noted that if the concentration of hyaluronidase in the sperm was too low, cervical packing with the enzyme increased considerably the number of successful fertilizations.

Very recent observations point to the fact that the spreading effect of the hyaluronidase may also prove valuable in certain roentgenologic applications. Already in one of the early experiments it was demonstrated by Hoffman and Duran-Reynals[12] that the enzyme possesses the faculty of diffusing the intradermally injected india ink over a comparatively larger area than would occur with similar suspensions in normal saline. In the animals receiving india ink mixed with the testis extract the ink particles were widely distributed, gradually thinning out toward the edges. In the control animals the ink particles stopped suddenly at a short distance from the point of injection. It was noted that the spreading induced by the testis extract occurred mainly through the intercellular spaces.

Simon and Narins[13] in order to establish the absorption-promoting effect of hyaluronidase in connective tissue injected radiopaque substances subcutaneously and determined by means of serial roentgenograms the time of disappearance of these substances. The experiments were performed in guinea pigs. Four sites were injected in each animal, one with 12 TRU* of hyaluronidase (dissolved in 1 cc. of normal saline), another with 50 TRU of hyaluronidase and the remaining two with only 1 cc. normal saline for control. Test and control sites were rotated in a clockwise manner in successive animals. Twenty minutes later all sites were reinjected with 1 cc. of a radiopaque substance, consisting either of neoiopax or diodrast. According to the authors this interval of twenty minutes is important since no hastening of the absorption is observed if the enzyme and the radiopaque substances are injected together. Serial roentgenograms were then made every ten to twenty minutes. It was found that the mean time of the disappearance of the opaque substance from the control sites amounted to 113 minutes whereas from the test sites it was only seventy-nine minutes. It was also noted that the urinary bladder was visualized sooner in those animals in which hyaluronidase was used to prepare the injection sites. The chief value of the contribution of Simon and Narins lies in the fact that it affords a roentgenographic method for the evaluation of the subcutaneous hyaluronidase activity.

Burket and György[14] published a rather comprehensive report on the clinical value of hyaluronidase. The enzyme was used in 123 infants and children for such procedures as hypodermoclysis, phthalein excretion test and urography. A standard dose of 0.8 mg. of the dry substance† dissolved in 1 ml. of distilled water was given in every case. It was definitely established that the clysis solution plus hyaluronidase was absorbed considerably faster than the plain saline-glucose solution without the enzyme. The acceleration amounted to about 40 per cent and in many instances to more than this. No significant difference was noted, however, in the dye excretion or total renal fluid excretion when the enzyme was added to the phthalein excretion test.

The effect of hyaluronidase on urography

[12] Hoffman, D. C., and Duran-Reynals, F. The influence of testicle extract on the intradermal spread of injected fluids and particles. *J. Exper. Med.*, 1931, *53*, 387–398.

[13] Simon, N., and Narins, L. The effect of hyaluronidase on the absorption of a subcutaneously deposited radiopaque substance. Am. J. Roentgenol. & Rad Therapy, 1949, *61*, 91–94.

* Turbidity reduction unit.

[14] Burket, L. C., and György, P. Clinical observations on the use of hyaluronidase. *Pediatrics*, 1949, *3*, 56–63.

† The material was prepared by Wyeth, Inc., under the trade name "Hydase."

was observed by Burket and György in nine cases with previous control urograms. The subcutaneous method according to Nesbit and Douglas[15] was used which necessitates the injection of 10 to 15 ml. of a 35 per cent diodrast solution diluted in 35 to 75 ml. of normal saline. When 0.8 mg. of hyaluronidase was injected subcutaneously before the dye was given it was possible to reduce the amount of the dye to one half. Burket and György state that an adequate visualization was obtained in both control and experimental urograms but these latter showed deeper contrast, better filling and sharper markings. There were no untoward reactions or complications which could be attributed to the effect of the enzyme.

Obviously, further exhaustive clinical investigations are necessary before all the possibilities of the use of hyaluronidase are explored. The initial results obtained are encouraging.

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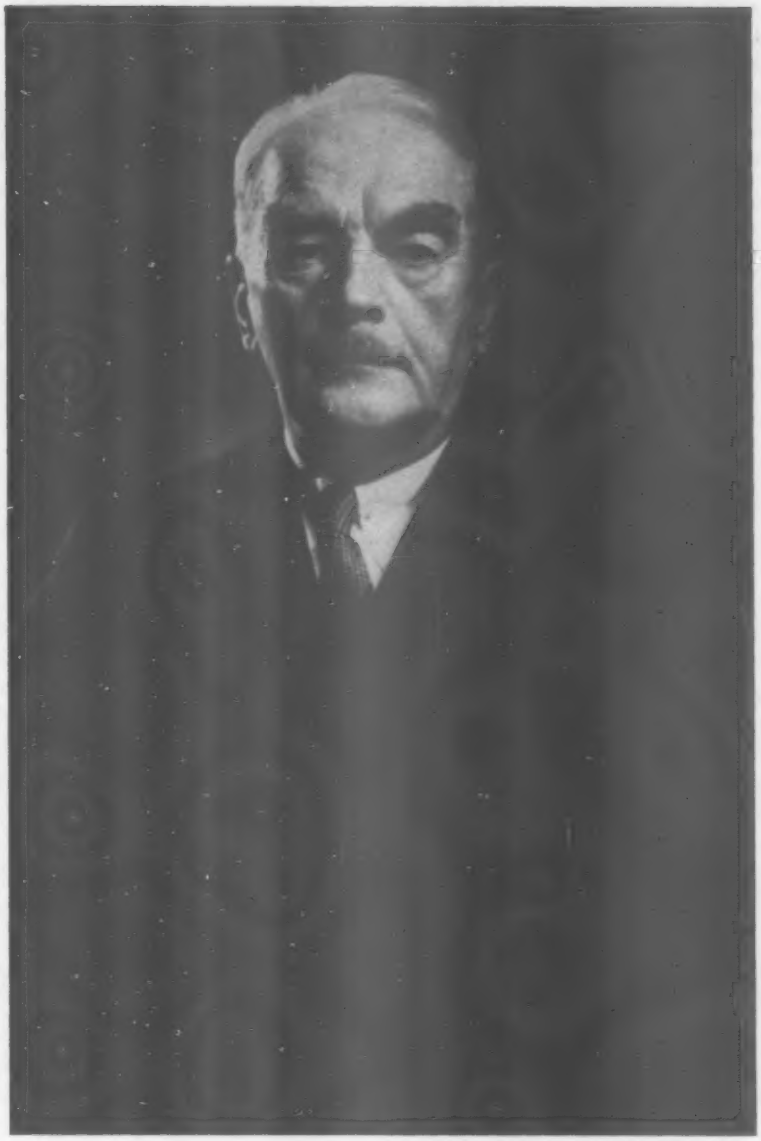

GORDON E. RICHARDS
1885-1949

ON THE evening of March 28, 1947, the late Dr. Gordon E. Richards, in Kingston, Ontario, for the official opening of the Pilot Cancer Clinic in that city, received the degree of Doctor of Laws at a special convocation of Queen's University. Dr. W. E. McNeill, Vice-Principal, presenting Dr. Richards to Dr. Wallace, said: "Mr. Vice Chancellor, in the name of the Senate of this University, I have the honour to present to you for the degree of Doctor of Laws, honoris causa, Gordon Earle Richards, Doctor of Medicine, Professor of Radiology at the University of Toronto, Di-

rector of the Department of Radiology at Toronto General Hospital, Managing Director of the Ontario Cancer Treatment and Research Foundation; a foremost Canadian scientist in cancer radiotherapy, internationally known as teacher, researcher, administrator, greatly serving mankind."

With brief eloquence the foregoing citation outlines the career of Dr. Richards, whose passing on the 13th of January, 1949, brought deep sorrow to his fellow-workers in the realm of Radiology in Canada, the United States and abroad. That he did greatly serve suffering mankind in one of its most grievous afflictions, the record of his more than thirty years of unremitting fight against cancer bears tribute. It was the grand aim and purpose of his life. Into that fight he threw all the zest of a crusader. All the knowledge, skill, originality and persistence with which he was so richly blessed, were devoted to that cause. It was the concentration of these qualities into his technique for the treatment of malignant disease that brought results of outstanding excellence and carried his fame abroad. He had an infinite capacity for detail and thoroughness. One example of this alone, the records of progress in countless numbers of cases treated, and illustrated with superb color photography, won admiration wide and sincere. His originality shone forth in the many special methods and devices he developed for the more efficient application of radium and roentgen rays in cancer treatment. In 1920, following studies in Britain and Germany, he established Canada's first high voltage center for cancer therapy, and every year the number of his patients extended into thousands. Some years ago he was the guest of honor at a dinner where one hundred women, all former cancer patients with no recurrence in periods from five to seventeen years, celebrated most happily.

But the price Dr. Richards paid for those years of service to others was the sacrifice of a serene old age in rural surroundings of which he had dreamed. In a confidential letter to a friend written in October, 1948, he spoke of the aplastic anemia due to radium from which he suffered, and he intimated that his remaining days on earth would be few. His death, three months later, removed a figure of world renown in the profession of Radiology, and he will be sadly missed by his co-workers in that field.

Gordon E. Richards, M.B.(Tor.), L.L.D. F.R.C.P.(C), F.A.C.R., and Honorary Member of the Royal Society of Medicine, England, was born at Lyn, Ontario, in 1885. His parents were the Reverend J. J. Richards and Anna Paul Richards. He attended public school in his native village and the Athens, Ontario, High School. Gold medallist in his year, he was graduated M.B. from the University of Toronto in 1908. Post-graduate studies were in Frankfurt, Germany, with Professors Dessauer and Holfelder, and later in London, Paris and Stockholm. His first practice was in British Columbia and included service as medical officer at a mining camp in the northerly reaches of that province. With the rank of captain he was Radiologist to No. 21 General Hospital, R.A.M.C., during the Dardanelles Campaign of the First World War, with duty on the Isle of Lemnos and at Alexandria, Egypt. Throughout the second world conflict he directed a Training School for Radiologists for the R.C.A.M.C. and the R.C.A.F.

It was in 1917 that he became the first Radiologist at Toronto General Hospital. His official positions included: Attending Radiologist-in-Chief, Toronto General Hospital; Director of Ontario Institute of Radiotherapy; Managing Director, Ontario Cancer Treatment and Research Foundation; Professor of Radiology, University of Toronto. His contributions to medical and radiological literature comprise a lengthy bibliography, and extended from 1916 to 1948. In 1947, Dr. Richards was selected to deliver the Skinner Lecture of the Faculty of Radiologists of London, on cancer of the breast. It was the first occasion since the lectureship was inaugurated that any

authority on cancer outside Europe was so honored. He was Founder of the Canadian Association of Radiologists, Fellow of the Toronto Academy of Medicine, member of the Ontario and Canadian Medical Associations, member of the American Roentgen Ray Society and of the Radiological Society of North America, and also Councillor of the American College of Radiology. He will be sadly missed by his fellow Radiologists, the profession at large and by a host of patients he has benefited.

A. C. Singleton, M.D.

RENÉ LEDOUX-LEBARD
1879-1948

THE American Roentgen Ray Society laments the passing of one of the outstanding radiologists of our time, whose loss will be universally felt. The influence of his work has been indelibly impressed on radiological practice both in the new world and in Europe.

René Ledoux-Lebard was born in Paris on April 21, 1879. His father, also a physician, was a former intern of the hospitals of Paris, chief of the Grancher Laboratory, and finally a member of the Pasteur Institute, where his chief work related to the bacillus of tuberculosis.

It was natural that René should follow in his father's footsteps Having completed his medical studies at the University of Paris, he served as extern with Gueyrat, Letulle and the internationally famous Antoine Béclère, who was for a generation the dean of radiologists of France, and a good friend of René's father. It was Béclère who inspired Ledoux-Lebard to take up the specialty of medical radiology. Ledoux-Lebard's thesis on "The Battle against Cancer," published in 1906 was said to be the first modern exposé on that question. It was also said that this published paper led to the foundation of the French Society for the Study of Cancer, of which he was secretary during many years.

As radiologist to the Salpêtrière he enjoyed with Gosset, Professor of Surgery at the University of Paris, a very warm and intimate relationship which continued for nearly forty years. Dr. Ledoux-Lebard made numerous researches in collaboration with Dauvillier in the field of radiation physics. It was due to his work with Dauvillier that he was able to provide some of the first apparatus for constant current deep radiotherapy He was a founder-member of the Society of Medical Radiologists of France, of which he was president in 1925, and one of the founders of the *Journal de radiologie et d'électrologie.*

During the first world war, 1914–1918, Dr. Ledoux-Lebard served as director of the radiological centers of the 9th Military Region at Tours, where in association with Ombredanne, the well-known surgeon, he carried out and published in book form a series of investigations on the localization and extraction of foreign bodies, a work which was translated into English and utilized very extensively by the British and American armies. He devised and had constructed several pieces of apparatus for military radiology which were adopted by the French army. As Senior Consultant in Radiology for the AEF, it was the privilege of the writer to arrange with Dr. Ledoux-Lebard in Tours for a school for the further instruction of officers of the United States Army Medical Corps, who had been sent to France as radiologists. Dr. Ledoux-Lebard gave generously of his time and of his friendship to one hundred or more American officers. As an aid to his English-speaking students he published a French-English dictionary of radiological terms. Also in 1921 he came to the United States as guest speaker of the American Roentgen Ray Society. It is understandable that Dr. Ledoux-Lebard should have enjoyed such a large circle of friends among the physicians of the United States.

In 1924 he was placed in charge of the course of Clinical Radiology of the Faculty of Medicine of Paris, which post he held for twenty-five years. He was a member of many French and foreign societies, including the American Roentgen Ray Society, before which Society he presented the Caldwell Lecture in 1921 and received honorary membership. He was also an honorary member of the Röntgen Society of London and delivered the Sylvanus Thompson Memorial Lecture in 1938; and he was also an honorary member of the Northern Society of Radiology, Stockholm.

His principal published works in book form included "Localisation et extraction des projectiles"; "Physiques des rayons X (with Dauvillier)"; his "Manuel de radiodiagnostic clinique," published in 1933, the second edition of which will appear in 1949; and his monumental "Technique du radiodiagnostic" with García-Calderon, published in 1943.

His works have dealt particularly with the relation of radiology to the localization and extraction of foreign bodies, the digestive tube, diseases of the liver, and of the bladder, certain osseous affections, and the gastric mucosa and urography (with Calderon). In radiotherapy he dealt mostly with the treatment of cancer. Altogether his published works have numbered about three hundred.

He was radiologist to the Franco-Musulman Hospital; consulting radiologist to the Ministry of Public Health, and of-

ficial radiologist to the Legion of Honor, and qualified as an expert witness before the tribunals. He was named Chevalier of the Legion of Honor in 1918 and an officer of the Legion in 1935.

Aside from his medical interests, Dr. Ledoux-Lebard was widely known as a

Medal of René Ledoux-Lebard

collector; and in his study on the technique and history of engravings in color and on the furniture of the XVIIIth Century, especially Empire furniture, he was an authority. He took great pride in his collection of richly bound books, and he also had great interest in ceramics.

The only modern author who was admitted to the honor of his hospitality was Anatole France, with whom he was well acquainted.

Dr. Ledoux-Lebard was recognized as a budgeteer of his time. He devoted his mornings to the examination and treatment of his hospitalized patients and to the teaching of roentgen diagnosis, in which he took pains not only to give theoretical lectures but also to add practical exercises in the interpretation of films by the students themselves. His afternoons he divided between his clientele, sales and libraries and antiques.

His relish for collecting did not diminish in any degree his speculative ambitions. It is told of him that he found under the screen in the stomach of a patient a silver spoon of ancient design, of which he himself had been robbed and which he had treasured greatly.

After becoming ill in January, 1947, he continued his work on his "Manuel de radiodiagnostic," in which he had the collaboration of his son, Dr. Guy Ledoux-Lebard. Death came on June 21, 1948.

Dr. Ledoux-Lebard's son, Guy, who has devoted years of serious study to general medicine, in the last few years has become a radiologist and now works with Professor Desgrez, the first Professor of Radiology, University of Paris, named two years ago, fifty years after Röntgen's discovery.

The colleagues and friends of Dr. René Ledoux-Lebard have struck off a medal, the execution of which was confided to Madame Yves Ledoux-Lebard, his daughter-in-law. His friends who may wish to procure a copy of this medal will receive one at the subscription price of fr. 800. Subscriptions may be sent by check or postal order in care of the Treasurer, M. Georges Masson, 120, Boulevard Saint Germain, Paris 6e, France.

James T. Case, M.D.

FRANK EDWARD SIMPSON
1868-1948

ON DECEMBER 13, 1948, Dr. Frank Edward Simpson passed away in Wesley Memorial Hospital, Chicago, ending the career of one long prominent in Chicago medicine.

Dr. Simpson was born in Saco, Maine on September 7, 1868, the son of Charles P. and Adelaide (Reade) Simpson. He received his A.B. degree from Bowdoin College in 1890 and his M.D. degree from Northwestern University Medical School, Chicago, in 1896. After interning at Cook County Hospital he was married to Miss Mary Beulah Lichty and began the prac-

tice of medicine, specializing in dermatology, in the year 1898, the same year the Curies discovered radium.

In 1910 he went abroad to study in Vienna and Paris. In Paris he saw the early clinical work with radium done by Dr. Regaud. In 1911 he returned to Chicago and brought the first radium to the city. He kept adding to his supply and in 1917 established an emanation plant in his office containing about 1.6 grams of radium. During these years he had been attending dermatologist to Cook County, Henrotin, Wesley, Alexin Brothers and the Chicago Policlinic Hospitals. He was also Adjunct Professor of Dermatology at Northwestern University Medical School.

He reported the first case of keratodermatitis gonorrheica to be diagnosed in America. In 1922 he published his book "Radium Therapy." In the same year he wrote the radium therapy section of the German textbook "Lehrbuch der Strahlentherapie". He also published a great number of articles on radium therapy in many American journals.

He was a charter member and one of the early presidents of the American Radium Society. He was also a member of the American College of Radiology, the American Medical Association, the Chicago Dermatological Society and the Chicago Roentgen Society.

J. Ernest Breed, M.D.

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this section solicited promptly after the events to which they refer

MEETINGS OF ROENTGEN SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. H. Dabney Kerr, University Hospital, Iowa City, Iowa. Annual meeting: Netherland Plaza Hotel, Cincinnati, Ohio, Oct. 4–7, 1949.

American Radium Society
Secretary, Dr. H. F. Hare, 605 Commonwealth Ave., Boston, Mass. Annual meeting: Ambassador Hotel, Atlantic City, N. J., June 5–7, 1949.

Radiological Society of North America
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y. Annual meeting: 1949, to be announced.

American College of Radiology
Executive Secretary, William C. Stronach, 20 N. Wacker Drive, Chicago 6. Annual meeting: Chalfonte-Haddon Hall, Atlantic City, N. J., June 5, 1949.

Section on Radiology, American Medical Association
Secretary, Dr. U. V. Portmann, Cleveland Clinic, Cleveland, Ohio, Annual Meeting: Atlantic City, N. J., June 8–10, 1949.

Alabama Radiological Society
Secretary, Dr. W. W. Anderson, Tuscaloosa, Ala. Next meeting time and place of Alabama State Medical Association, Montgomery, Ala., April 19–21, 1949.

Arkansas Radiological Society
Secretary, Dr. Fred Hames, 511 National Bldg., Pine Bluff, Ark. Meets every three months and also at time and place of State Medical Association.

Brooklyn Roentgen Ray Society
Secretary, Dr. A. H. Levy, 1354 Carroll St., Brooklyn 13, N. Y. Meets monthly on fourth Tuesday, October to April.

Buffalo Radiological Society
Secretary, Dr. Mario C. Gian, 610 Niagara St., Buffalo, N. Y. Meets second Monday evening each month, October to May inclusive.

Central New York Roentgen Ray Society
Secretary, Dr. Dwight V. Needham, 608 E. Genesee St., Syracuse N. Y. Meets January, May, November.

Central Ohio Radiological Society
Secretary, Dr. Paul D. Meyer, Grant Hospital, Columbus, Ohio. Meets at 6:30 P.M. on second Thursday of October, December, February, April, and June at Seneca Hotel, Columbus, Ohio.

Chicago Roentgen Society
Secretary, Dr. John H. Gilmore, 720 N. Michigan Ave., Chicago 11, Ill. Meets second Thursday of each month October to April inclusive at the Palmer House.

Cincinnati Radiological Society
Secretary, Dr. Eugene L. Saenger, 735 Doctors Bldg., Cincinnati 2, Ohio. Meets last Monday of each month, September to May, inclusive.

Cleveland Radiological Society
Secretary, Dr. Merthyn A. Thomas, 10515 Carnegie Ave. Cleveland 6, Ohio. Meetings at 6:30 P.M. on fourth Monday of each month from October to April.

Colorado Radiological Society
Secretary, Dr. Mark S. Donovan, 306 Majestic Bldg., Denver 2, Colo. Meets third Friday of each month at Department of Radiology, Colorado School of Medicine.

Connecticut Valley Radiologic Society
Secretary, Dr. E. W. Godfrey, 1676 Boulevard, West Hartford, Conn. Meets second Friday of October and April.

Dallas-Fort Worth Roentgen Study Club
Secretary, Dr. X. R. Hyde, Medical Arts Bldg., Fort Worth, Texas. Meets in Dallas on odd months and in Fort Worth on even months, on third Monday, 7:30 P.M.

Detroit Roentgen Ray and Radium Society
Secretary, Dr. W. G. Belanger, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne County Medical Society Building.

East Bay Roentgen Society
Secretary, Dr. Dan Tucker, 434-30th St., Oakland 9, Calif. Meets first Thursday each month at Peralta Hospital, Oakland.

Florida Radiological Society
Secretary, Dr. F. K. Hurt, Riverside Hospital, Jacksonville, Fla. Meets twice annually, in the spring with the annual State Society meeting, and in the fall.

Georgia Radiological Society
Secretary, Dr. Robert Drane, DeRenne Apartments, Savannah, Ga. Meets in mid-winter and at annual meeting of Medical Association of Georgia in the spring.

Houston X-ray Club
Secretary, Dr. Curtis H. Burge, 3020 San Jacinto St., Houston 4, Texas. Meets fourth Monday each month.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.

Illinois Radiological Society
Secretary, Dr. Wm. DeHollander, St. John's Hospital, Springfield, Ill. Meets three times a year.

Indiana Roentgen Society
Secretary, Dr. William M. Loehr, 712 Hume-Mansur Bldg., Indianapolis 4. Meets second Sunday in May.

Iowa X-Ray Club
Secretary, Dr. Arthur W. Erskine, 326 Higley Bldg., Cedar Rapids, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. Special meetings by announcement.

Kansas Radiological Society
Secretary, Dr. Anthony F. Rossitto, Wichita Hospital, Wichita, Kan. Meets annually with State Medical Society.

Kentucky Radiological Society
Secretary, Dr. W. C. Martin, 321 W. Broadway, Louisville. Meets annually in Louisville on first Saturday in Apr.

Long Island Radiological Society
Secretary, Dr. Marcus Wiener, 1430-48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Los Angeles Radiological Society
Secretary, Dr. Wybren Hiemstra, 1414 S. Hope St., Los Angeles 15, Calif. Meets second Wednesday each month at Los Angeles County Medical Assn. Building.

Louisiana Radiological Society
Secretary, Dr. J. R. Anderson, 1130 Louisiana Ave., Shreveport. Meets annually during Louisiana State Medical Society Meeting.

Louisville Radiological Society
Secretary, Dr. E. L. Pirkey, Louisville General Hospital, Louisville 2, Ky. Meets monthly on second Friday at Louisville General Hospital.

* Secretaries of societies not here listed are requested to send the necessary information to the Editor.

Michigan Association of Roentgenologists
Secretary, Dr. R. D. McDuff, 220 Genesee Bank Bldg., Flint 3, Mich.

Milwaukee Roentgen Ray Society
Secretary, Dr. C. A. H. Fortier, 231 W. Wisconsin Ave., Milwaukee, Wis. Meets monthly on second Monday at University Club.

Minnesota Radiological Society
Secretary, Dr. Chauncey N. Borman, 802 Medical Arts Bldg., Minneapolis 2, Minn. Two meetings yearly, one at time of Minnesota State Medical Association the other in the fall.

Nebraska Radiological Society
Secretary, Dr. Ralph C. Moore, Nebraska Methodist Hospital, Omaha 3, Nebr. Meets third Wednesday of each month, at 6 P.M. at either Omaha or Lincoln.

New England Roentgen Ray Society
Secretary, Dr. George Levene, Massachusetts Memorial Hospitals, Boston, Mass. Meets monthly on third Friday, Boston Medical Library.

New Hampshire Roentgen Ray Society
Secretary, Dr. A. C. Johnston, Elliott Community Hospital, Keene, N. H. Meets four to six times yearly.

New York Roentgen Society
Secretary, Dr. Ramsay Spillman, 115 East 61st St., New York City. Meets monthly on third Monday, New York Academy of Medicine, at 8:30 P.M.

North Carolina Radiological Society
Secretary, Dr. J. E. Hemphill, 323 Professional Bldg., Charlotte 2, N. C. Meets in May and October.

North Dakota Radiological Society
Secretary, Dr. C. O. Heilman, 807 Broadway, Fargo. Meetings held by announcement.

Northern California Radiological Club
Secretary, Dr. C. E. Grayson, Medico-Dental Bldg., Sacramento 14, Calif. Meets at dinner last Monday, every second month, except June, July and August.

Ohio State Radiological Society
Secretary, Dr. Carroll C. Dundon, 2065 Adelbert Road, Cleveland 6, Ohio.

Oklahoma State Radiological Society
Secretary, Dr. W. E. Brown, Tulsa, Okla. Three regular meetings annually.

Oregon Radiological Society
Secretary, Dr. Boyd Isenhart, 214 Medical Dental Bldg., Portland 5, Oregon. Meets monthly 2nd Wednesday, 8:00 P.M., Library of University of Oregon Medical School.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets first Tuesday of each month.

Pacific Northwest Radiological Society
Secretary, Dr. S. J. Hawley, 1320 Madison St., Seattle 4, Wash. Meets annually in May.

Pacific Roentgen Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. J. M. Converse, 416 Pine St., Williamsport. Annual Meeting: May 20 and 21, 1949, Bedford Springs Hotel, Bedford, Pa.

Philadelphia Roentgen Ray Society
Secretary, Dr. Arthur Finkelstein, Graduate Hospital, 19th and Lombard St. Meets first Thursday each month October to May, at 8:00 P.M., in Thomson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. R. P. Meader, 4002 Jenkins Arcade Pittsburgh 22, Pa. Meets 6:30 P.M. at Webster Hall Hotel on second Wednesday each month, October to May inclusive.

Queens Roentgen Ray Society
Secretary, Dr. J. E. Goldstein, 88-29 163rd St., Jamaica 3, N. Y. Meets fourth Monday of each month except during the summer.

Radiological Section, Baltimore Medical Society
Secretary, Dr. Harry A. Miller, 2452 Eutaw Place, Baltimore. Meets third Tuesday each month, September to May.

Radiological Section, Connecticut Medical Society
Secretary, Dr. Robert M. Lowman, Grace-New Haven Community Hospital, New Haven 11. Meets bimonthly second Thursday, at place selected by Secretary.

Radiological Section, District of Columbia Medical Society
Secretary, Dr. A. A. J. Den, 1801 K St., N. W., Washington, D. C. Meets Medical Society Auditorium, third Thursday, January, March, May, October at 8:00 P.M.

Radiological Section, Southern Medical Association
Secretary, Dr. Roy G. Giles, Temple, Texas.

Radiological Society of New Jersey
Secretary, Dr. Raphael Pomeranz, 31 Lincoln Park, Newark, N. J. Meets annually at time and place of State Medical Society. Mid-year meetings at place chosen by president.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Ralph E. Alexander, 101 Medical Arts Bldg. Meets monthly on third Monday from October to May, inclusive, 8 P.M. at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary, Dr. Maurice D. Frazer, 1037 Stuart Bldg., Lincoln, Nebr. Meets Denver, Colo., August 18, 19, 20, 1949.

St. Louis Society of Radiologists
Secretary, Dr. Edwin C. Ernst, Beaumont Medical Building, St. Louis, Mo. Meets fourth Wednesday of each month, except June, July, August, and September.

San Diego Roentgen Society
Secretary, Dr. R. F. Niehaus, 1831 Fourth Ave., San Diego, Calif. Meets monthly, first Wednesday at dinner.

Section on Radiology, California Medical Association
Secretary, Dr. D. R. MacColl, 2007 Wilshire Blvd., Los Angeles 5, Calif.

Section on Radiology, Illinois State Medical Society
Secretary, Dr. Harold L. Shinall, St. Joseph's Hospital, Bloomington, Ill.

Shreveport Radiological Club
Secretary, Dr. R. W. Cooper, Charity Hospital, Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M., September to May inclusive.

South Carolina X-Ray Society
Secretary, Dr. T. A. Pitts, Baptist Hospital, Columbia, S. C. Meets in Charleston on first Thursday in November, also at the time and place of South Carolina State Medical Association.

Tennessee Radiological Society
Secretary, Dr. J. M. Frère, 707 Walnut St., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

Texas Radiological Society
Secretary, Dr. R. P. O'Bannon, 650 Fifth Ave., Fort Worth 4, Texas.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

University of Wisconsin Radiological Conference
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets first and third Thursdays 4:00 to 5:00 P.M., September to May inclusive. Room 203, Service Memorial Institute, 426 N. Charter St., Madison.

Utah State Radiological Society
Secretary, Dr. Angus K. Wilson, 343 S. Main St., Salt Lake City 1, Utah. Meets third Wednesday in September, November, January, March and May.

Virginia Radiological Society
Secretary, Dr. P. B. Parsons, Norfolk General Hospital, Norfolk, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. Homer V. Hartzell, 310 Stimson Bldg., Seattle 1, Wash. Meets fourth Monday each month, October through May, College Club, Seattle.

X-Ray Study Club of San Francisco
Secretary, Dr. Ivan J. Miller, 49 Fourth St., San Francisco 3. Meets monthly on third Thursday at 7:45 P.M., January to June at Lane Hall, Stanford University Hospital, and July to December at Langley Porter Clinic, University of California Hospital.

Cuba

Sociedad de Radiología y Fisioterapia de Cuba
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

Mexico

Sociedad Mexicana de Radiologia y Fisioterapia
General Secretary, Dr. D. P. Cossio, Marsella No. 11, Mexico, D. F. Meets first Monday of each month.

British Empire

British Institute of Radiology Incorporated with the Röntgen Society
Ordinary meeting, on the Thursday preceding the third Friday, October to May at 8:15 P.M.
Medical Members' meeting, on third Friday in each month at 5:00 P.M., 32 Welbeck St., London, W 1.

Faculty of Radiologists
Honorary Secretary, Dr. J. F. Bromley, 45, Lincoln's Inn Fields, London, W.C.2, England.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London.

Canadian Association of Radiologists
Honorary Secretary, Dr. E. M. Crawford, 2100 Marlowe Ave., Montreal 28, Que. Meetings January and June.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Société Canadienne-Francaise d'Electrologie et de Radiologie Médicales
Secretary, Dr. Origéne Dufresne, 4120 Ontario St., East, Montreal, P. Q.

Australian and New Zealand Association of Radiologists
Honorary Secretary, Dr. Alan R. Colwell, 135 Macquarie St., Sydney, N.S.W.
Honorary Secretaries, State Branches:
New South Wales, Dr. E. W. Frecker, 135 Macquarie St., Sydney.
Victoria, Dr. T. L. Tyrer, 3 Lockerbie Court, East St. Kilda.
Queensland, Dr. J. Adam, 131 Wickham Terrace, Brisbane.
South Australia, Dr. B. C. Smeaton, 178 North Terrace, Adelaide.
Western Australia, Dr. A. M. Nelson, 179-B St. Georges Terrace, Perth.
New Zealand, Dr. E. G. Lynch, 12 Bolton St., Wellington.

South America

Sociedad Argentina de Radiologia
Secretary, Dr. Guido Gotta, Buenos Aires, Argentina. Meetings are held monthly.

Sociedade Brasileira de Radiologia Medica
Secretary, Dr. Nicola Caminha, Av. Mem de Sa, Rio de Janeiro, Brazil. Meets monthly, except during January, February and March.

Sociedade Brasileira de Radioterapia
Secretary, Dr. Andrelino Amaral, Av. Brigadeiro Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Tuesday at 9 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Peruana de Radiologia
Secretary, Dr. Julio Bedoya Paredes, Apartado, 2306, Lima, Peru. Meets monthly except during January, February and March, at Asociación Médica Peruana "Daniel A. Carrión," Villalta, 218, Lima.

Sociedad de Radiologica, Cancerologia y Fisica Medica del Uruguay
Secretary, Dr. Arias Bellini.

Continental Europe

Société Belge de Radiologie
General Secretary, Dr. S. Masy, 111 Avenue des Alliés, Louvain, Belgium. Meets monthly, second Sunday at Maison des Médecins, Brussels.

Ceskoslovenská společnost pro röntgenologii a radiologii v Praze
Secretary, Dr. Roman Bláha, Praha XII, stát. nemocnice, Czechoslovakia. Meets monthly except during July, August, and September. Annual general meeting.

Polish Society of Radiology
Secretary, Dr. L. Zgliczynski, 59 Nowogrodzka St., Warsaw, Poland. Next meeting, Krakow, June 2 and 3, 1949.

Gdansk Section, Polish Society of Radiology
Secretary, Dr. A. Smigielska, Akademia Lekarska, Gdansk. Meets monthly last Sunday at 10.30, X-Ray Dept., Akademia Gdansk.

Warsaw Section, Polish Society of Radiology
Secretary, Dr. L. Zgliczynski, 59 Nowogrodzka St., Warsaw, Poland. Meets monthly.

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banual Mărăcine, 30, S. I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad.
USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

Leningrad Roentgen Ray Society
Secretaries, Drs. S. G. Simonson and G. A. Gusterin. Meets monthly, first Monday at 8 o'clock, State Institute of Roentgenology and Radiology, Leningrad.

Moscow Roentgen Ray Society
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky. Meets monthly, first Monday, 8 P.M.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

Sociedad Espanola de Radiologia y Electrologia
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7. Madrid, Spain. Meets monthly in Madrid.

Schweizerische Röntgen-Gesellschaft (Société Suisse de Radiologie)
President, Dr. H. E. Walther, Gloriastr. 14, Zürich, Switzerland.

Societa Italiana di Radiologia Medica
Secretary, Prof. Mario Ponzio, Ospedale Mauriziano Torino, Italy. Meets biannually.

CANCER TEACHING DAY

A Cancer Teaching Day was held at the Hermann M. Biggs Memorial Hospital, Ithaca, New York, on January 19, 1949. This was presented under the auspices of the Medical Society of the County of Tompkins, the Medical Society of the State of New York, Tompkins County Department of Health, and the New York State Department of Health, Bureau of Cancer Control.

The Chairman of the afternoon meeting, which was called to order at four o'clock, was Dr. N. Stanley Lincoln, Director, Hermann M. Biggs Memorial Hospital. The following papers were presented:

Cancer of the Breast. By Dr. Norman Treves, Associate Attending Surgeon, Memorial Hospital, New York.

Operative Risk and Preoperative and Postoperative Care of Patients with Cancer. By Dr. John S. LaDue, Associate Attending Physician, Memorial Hospital, New York.

The Chairman of the evening meeting was Dr. C. Stewart Wallace, President, Medical Society, County of Tompkins. The two following papers were given:

Significance and Management of Abnormal Vaginal Bleeding. By Dr. Clyde L. Randall, Professor Gynecology, University of Buffalo School of Medicine, Buffalo, N.Y.

Gastrointestinal Cancer. By Dr. Albert F. R. Andresen, Professor of Clinical Medicine, Long Island College of Medicine, Brooklyn, N. Y.

CONFERENCE OF HEALTH OFFICERS AND PUBLIC HEALTH NURSES

Lake Placid, New York, has been chosen by the New York State Department of Health and the State Health Officers Association as the location for the Forty-fifth Annual Conference of Health Officers and Public Health Nurses. The conference dates will be June 20–23, 1949, as announced by Dr. Herman E. Hilleboe, New York State Commissioner of Health. The Association of School Physicians will hold its annual meeting on the opening day, in conjunction with the general conference of public health administrators. Clifford M. Hodges, press representative of the State Department of Health, has been appointed as conference manager.

This meeting, one of the oldest and best known in the country, annually attracts the nation's leaders in the field of public health as speakers and participants.

SAMUEL FREEDMAN LECTURES

On Saturday and Sunday, April 30 and May 1, 1949, Dr. LeRoy Sante, Professor of Roentgenology at St. Louis University, St. Louis, Missouri, will deliver the first annual Samuel Freedman Lectures on Diagnostic Roentgenology at the University of Cincinnati College of Medicine. Roentgenologists desiring to attend are requested to write Dr. Benjamin Felson, X-ray Department, Cincinnati General Hospital, Cincinnati, Ohio, for further details.

PENNSYLVANIA RADIOLOGICAL SOCIETY

The Pennsylvania Radiological Society will hold its Thirty-fourth Annual Meeting on Friday and Saturday, May 20 and 21, 1949, at the Bedford Springs Hotel, Bedford, Pennsylvania. Dr. Leslie H. Osmond of Pittsburgh, President, will have charge of an interesting scientific program arranged by the Program Chairman, Dr. Ralph D. Bacon, Erie, and his committee.

THIRD INTER-AMERICAN CONGRESS OF RADIOLOGY

For information concerning the Third Inter-American Congress of Radiology to be held in Santiago, Chile, November 11–17, 1949, previous issues of the JOURNAL may be consulted, particularly the October, 1948, issue, page 559, the January, 1949, issue, page 116, and the February, 1949, issue page 257.

SIXTH INTERNATIONAL CONGRESS OF RADIOLOGY

The Sixth International Congress of Radiology is to be held in London, England, from July 23 to 30, 1950, with Congress headquarters at the Central Hall, Westminster. Information concerning this Congress was published in the February, 1949, issue of the JOURNAL, page 257.

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

Arthritis and Related Conditions. Edited by Theodore Franklin Bach, M.D., F.A.C.P., Assistant Professor of Medicine in the Graduate School of Medicine of the University of Pennsylvania, Philadelphia; Chief of Arthritis Clinic, Abington Memorial Hospital, Abington, Pa. Cloth. Price, $6.50. Pp. 452, with 146 illustrations. Philadelphia: F. A. Davis Company, 1948.

The plans and production of this book were first organized by Dr. R. Garfield Snyder, whose untimely death cut short his careful planning for its publication. The publisher, at the suggestion of Dr. George Morris Piersol, asked Dr. Bach to re-evaluate and complete the task.

In addition to the work of Drs. Snyder and Bach, contributions were made by Irvin Balensweig, M.D., William Bates, M.D., J. Borak, M.D., Edith H. Brokaw, O.T.R., K. G. Hansson, M.D., Arthur Krida, M.D., John C. McCauley, Jr., M.D., Dudley J. Morton, M.D., C. H. Traeger, M.D. Each of these authors has contributed especially with regard to the treatment of arthritis and each is a specialist in his or her own field.

The book has been written primarily for the general practitioner but it gives to the radiologist the advantages of knowing what is of special interest to the general practitioner in managing arthritis, according to various points of view of these specialists, in association with or independent of radiology.

The book comprises twenty-eight chapters, and fourteen of the last eighteen have been written by the authors named above who have taken interest in particular phases of arthritis, but especially with regard to therapy. The broad view taken by the Editor, Dr. Bach, regarding arthritis is in part surely due to his own association with Dr. Ralph Pemberton, one of our greatest authorities on rheumatism. His broad view is further illustrated by his statement regarding the treatment of rheumatoid arthritis, Chapter ix, in which he says, "Since we are dealing with a disease of unknown etiology, the treatment cannot be stereotyped."

Of special interest to the radiologist are Chapters xiii and xiv, written by J. Borak M.D., and these chapters include 54 pages of the book. During eight years, Dr. Borak had been Director of the Department of Roentgen Therapy in the Institute of Dr. Guido Holzknecht in Vienna. Subsequently he served for eight years as Director of the Radiological Department in the Rothschild Hospital in Vienna, where the roentgen rays came to be considered an essential in the treatment of arthritis, as it has long been recognized to be in the fields of tumors and skin diseases. During recent years, he has been doing roentgen therapy in arthritic conditions in the Clinic of the Medical College of New York University, and in the Goldwater Memorial Hospital, New York City.

Borak discusses in detail the pathological conditions in the tissues involved in arthritis, and then under "Principles of X-ray Therapy," he indicates the varations in the technique that are necessary to adapt the radiation effects to the pathological conditions that are present in the particular individual case under treatment. He indicates that the results to be obtained from roentgen therapy will vary in great part with the knowledge possessed by the radiologist regarding the conditions in the joints affected, and the skill with which the radiologist adapts the treatment to the condition of the tissue present in the joints. These principles discussed by Dr. Borak are of course those which are involved in any roentgen therapy. Too often failures or poor results in the past have occurred from the lack of proper technique.

In the diagnosis of the pathological conditions in arthritis, Drs. Pemberton and Bach have made special use of studies by means of the roentgen rays in the hands of various experts in Philadelphia.

In connection with the chapters on the various methods of therapy of arthritis, each author lists the bibliography giving the most important references bearing upon the subject under discussion. One is impressed, therefore, with the thoroughness with which this whole book is treated. This book should be in every radiologist's library as well as in that of every practitioner who is dealing with arthritis. The

discussion by Dr. Borak seems to indicate that roentgen therapy has not been sufficiently or properly evaluated in connection with the therapy of arthritis.

George E. Pfahler, M.D.

Essentials of Pathology. By Lawrence W. Smith, M.D., F.C.A.P., Formerly Professor of Pathology, Temple University School of Medicine; Associate Professor of Pathology, Cornell University Medical School, and Assistant Professor of Pathology, Harvard Medical College, etc., and Edwin S. Gault, M.D., F.C.A.P., Associate Professor of Pathology and Bacteriology, Temple University School of Medicine. With a foreword by the late James Ewing, M.D., Memorial Hospital, New York City. Third edition. Cloth. Price, $12.00. Pp. 764, with 740 illustrations, a number in color. Philadelphia: The Blakiston Company, 1948.

Due to the rapidly increasing complexity of pathology the presentation of both the fundamental principles and the entire gamut of disease entities in one useful volume has become an inpossible task fraught with confusion. Therefore it is obvious that a thorough understanding of basic principles is an absolute necessity for students and practitioners alike. Such knowledge is equally essential for a proper understanding of the more complex encyclopedic presentations of disease and for use in connection with everyday experiences with morbid processes.

With this concept in mind the authors of Essentials of Pathology are to be commended for limiting the subject matter to fundamental principles, enlivened by the frequent use of complete case histories, photographs and reproductions of roentgenograms.

The use of illustrative case histories is one of the oldest and best methods of teaching, but its utilization in textbooks of pathology is novel. There can be no doubt that the case histories together with the fine photographs accomplish much in preventing the boredom which usually accompanies consideration of fundamental facts alone. However, the fundamentals are presented in a refreshingly clear and concise manner at the beginning of each subject under consideration. This material is followed, in many instances, by carefully chosen case histories which include thorough descriptions of gross and microscopic findings. The frequent use of excellent photographs, photomicrographs and roentgenograms gives visual assistance in developing the relationship between gross and microscopic changes and the clinical picture. Conversely, this clinicopathological correlation is an impressive aid in retaining and using the basic facts.

No attempt has been made to cover the entire field of pathology. Many of the less important disease entities are omitted. Controversial subjects are presented with appropriate brevity. The material is arranged into the familiar divisions of general pathology and systemic pathology for obvious reasons. A relatively large amount of space is devoted to consideration of neoplasms. This was done because of the relative increasing incidence of cancer and of the tremendous interest in the entire group of neoplastic diseases. Considerable space is also devoted to parasitic diseases because worldwide travel may increase the incidence of these entities.

The third edition is a more complete textbook than its predecessors. Its contents include many of the recent advances in pathology. The book has been completely revised. The page size has been reduced, facilitating handling and storing. The pages have double columns of easily readable print.

The reviewer recommends this book for students who are entering the field of medicine and for postgraduate students who for various reasons wish a review of pathology.

D. L. Hinerman, M.D.

A Symposium on the Use of Isotopes in Biology and Medicine. Addresses delivered at an Institute held at the University of Wisconsin from September 10 to 13, 1947. Cloth. Price, $5.00. Pp. 445, with illustrations. Madison, Wisconsin: University of Wisconsin Press, 1948.

The University of Wisconsin, in September, 1947, sponsored the symposium whose proceedings are reported in this volume. The twenty chapters, each by a recognized authority in his branch of the subject, furnish an authoritative text on tracer uses and techniques of isotopes, with less detailed data on therapeutic procedures and safety measures.

The symposium is opened by Hans Clark with a brief account of the history of isotopes in biochemistry. Preparation of stable isotopes is then discussed by Harold Urey, and of radioactive isotopes by Glenn Seaborg. This section is completed by the presentation by Paul

Aebersold of information regarding the availability of isotopes and their distribution by the Atomic Energy Commission.

Then follow four chapters on various technical topics: detection of stable isotopes, by Alfred Nier, detection and measurement of radioactivity, by Charles Coryell, assay of radioactive isotopes in biological research, by Martin Kamen, and preparation of compounds containing isotopes, by Donald Melville.

After these basic presentations come a series of discussions of information obtained by the uses of tracers. First are covered four biochemical topics: protein metabolism, by David Sprinson, intermediary carbohydrate metabolism, by Harland Wood, intermediary metabolism of lipids, by Konrad Bloch, and metabolism of mineral elements, by David Greenberg. Several clinical applications are then outlined: iodine metabolism and thyroid function, by I. L. Chaikoff and A. Taurog, medical applications of tracers, by Joseph Hamilton, therapeutic uses of radiophosphorus, by Byron Hall, and treatment of thyroid disease with radioiodine, by Saul Hertz.

Health hazards from isotopes and protection against them are topics of chapters by William Bale and James Nickson, the former presenting the special hazards and the latter giving information regarding their avoidance.

The book ends with two essays by Harold Urey and Farrington Daniels, on international aspects of atomic energy, and on the development of atomic energy, giving some expression of the scientists' attitude toward the political and sociological implications of the atomic energy problem.

As in any compendium, some chapters stand out as better organized or more clearly presented than others, but the overall standard is high. The book was lithoprinted to speed production, and is perhaps not as pleasing to the eye as standard print, but the legibility has not suffered. Because of the method of publication. the authors did not see proof, and a number of errors crept in, mostly unimportant. These have, however, all been collected on a single errata sheet.

The book is a very useful addition to the library of anyone concerned with biological, physiological, or medical uses of stable or radioactive isotopes, especially in the field of tracer methodology.

Edith H. Quimby

BOOKS RECEIVED

Management of Common Gastro-intestinal Diseases. Edited by Thomas A. Johnson. The American Practitioner Series. Cloth. Price, $7.00. Pp. 280, with 16 illustrations. Philadelphia: J. B. Lippincott Co., 1948.

Acute Intestinal Obstruction. By Rodney Smith, M.S., F.R.C.S., Assistant Surgeon, St. George's Hospital, London, Consulting Surgeon, Wimbledon Hospital; Hunterian Professor, Royal College of Surgeons. With a chapter on Radiological Diagnosis, by Eric Samuel, M.D., F.R.C.S., F.F.R., D.M.R.E., Late Radiologist, The Middlesex Hospital, London. Foreword by Rupert Vaughan Hudson, F.R.C.S. Cloth. Price, $5.00. Pp. 259, with 102 illustrations. Baltimore: The Williams and Wilkins Co., 1948.

Clinical Roentgenology of the Digestive Tract. By Maurice Feldman, M.D., Assistant Professor of Gastroenterology, University of Maryland; Associate in Gastroenterology, Mercy Hospital; Consulting Roentgenologist, Sinai Hospital. Third edition. Cloth. Price, $8.00. Pp. 901, with 641 illustrations. Baltimore: The Williams and Wilkins Co., 1948.

The 1948 Year Book of Radiology. *Diagnosis*. Edited by Fred Jenner Hodges, M.D., Professor and Chairman, Department of Roentgenology, University of Michigan. *Therapeutics*. Edited by Ira I. Kaplan, M.D., F.A.C.R., Director, Radiation Therapy Department, Bellevue Hospital, New York City; Clinical Professor of Radiology, New York University Medical College, etc. Cloth. Price, $6.50. Pp. 472, with 393 illustrations. Chicago: Year Book Publishers, Inc., 1948.

The Skull, Sinuses and Mastoids: A Handbook of Roentgen Diagnosis. By Barton R. Young, M.D., Professor of Radiology, Temple University Medical School. Cloth. Price, $6.50. Pp. 328, with 141 illustrations. Chicago: Year Book Publishers, 1948.

Malignant Disease and its Treatment by Radium. By Sir Stanford Cade, K.B.E., C.B., F.R.C.S., M.R.C.P., Surgeon, Westminster Hospital, Mount Vernon Hospital and Radium Institute; Lecturer in Surgery, Westminster Hospital Medical School and formerly Examiner in Surgery, University of London, etc. With a Foreword by Sir Ernest Rock Carling, F.R.C.P., F.R.C.S., F.F.R., Consulting Surgeon and Vice-President,

Westminster Hospital. Second edition. Vol. I. Cloth. Price, $12.50. Pp. 383, with 161 illustrations. Baltimore: The Williams and Wilkins Co., 1948.

The Treatment of Malignant Disease by Radium and X-Rays: Being a Practice of Radiotherapy. By Ralston Paterson, M.C., M.D., F.R.C.S.E., D.M.R.E., F.F.R., Christie Hospital and Holt Radium Institute, Manchester. Cloth. Pp. 622, with numerous illustrations. Baltimore: The Williams and Wilkins Co., 1948.

Radon: Its Technique and Use. By W. A. Jennings, B.Sc., A. Inst. P., Physicist to the Royal Northern Hospital and Prince of Wales's General Hospital, Joint Radiotherapy Centre, and S. Russ, C.B.E., D.Sc., F. Inst. P., Formerly Physicist to the Middlesex Hospital, London. Cloth. Price, 18s. Pp. 222, with 12 pages of half-tones and 49 figures. London: John Murray, 1948.

Die Indikationen zur Röntgen- und Radiumbestrahlung. Von Dr. Med. Habil. R. Glauner, Dozent für Röntgenologie und Strahlenheilkunde, Stuttgart. Paper. Price, DM 7.20. Pp. 128. Stuttgart: Georg Thieme Verlag, 1948.

The Thyroid and Its Diseases. By J. H. Means, M.D., Jackson Professor of Clinical Medicine, Harvard University, and Chief of the Medical Services, Massachusetts General Hospital. Second edition. Cloth. Price. $12.00. Pp. 571, with 63 illustrations. Philadelphia: J. B. Lippincott Co., 1948.

Essentials of Pathology. By Lawrence W. Smith, M.D., F.C.A.P., Formerly Professor of Pathology, Temple University School of Medicine; Associate Professor of Pathology, Cornell University Medical School, and Assistant Professor of Pathology, Harvard Medical College, etc., and Edwin S. Gault, M.D., F.C.A.P., Associate Professor of Pathology and Bacteriology, Temple University School of Medicine. With a foreword by the late James Ewing, M.D., Memorial Hospital, New York City. Third edition. Cloth. Price, $12.00. Pp. 764, with 740 illustrations, a number in color. Philadelphia: The Blakiston Co., 1948.

A Symposium on the Use of Isotopes in Biology and Medicine. Addresses delivered at an Institute held at the University of Wisconsin from September 10 to 13, 1947. Cloth. Price, $5.00. Pp. 445, with illustrations. Madison, Wisconsin: University of Wisconsin Press, 1948.

Electromagnetic Waves and Light: An Introductory Physical Discussion. First Part. By Charles F. Meyer, Associate Professor of Physics, University of Michigan. Paper. Price, $1.35. Pp. 83, with illustrations. Ann Arbor, Michigan: Edwards Brothers, Inc., 1948.

Vol. 61, No. 3

ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

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ROENTGEN DIAGNOSIS

HEAD

Engeset, Arne, and Kvadsheim, Hans. Technical improvements in cerebral angiography. *Acta radiol.*, January, 1948, *29*, 83–86.

The authors describe an apparatus for automatically exposing films in the process of arteriography. They added a system comprising an electric contact with attachment to the injection syringe combined with an attachment of an electric timer which turns on the roentgen rays at any given time, from one to ten seconds after the exposure of the arteriogram. In this way, phlebograms can be obtained at the same time as the arteriogram.

For angiography, they use a Lysholm-Schonander skull table with the supplementary cassette magazine for two films. A small lamp showing light during each exposure is mounted on the electric timer to serve as a signal to the assistant who is to pull away the first exposed film (the arteriogram) after which the second cassette automatically comes into position for the second exposure (the phlebogram).

This new technique has been used successfully in over one hundred examinations.

They have used the electric contact attachment to the syringe of Zehnder which turned on the roentgen rays at any given position of the piston.—*Bernard Halper, M.D.*

Wechsler, S., and Gross, S. W. Cerebral arteriography in subarachnoid hemorrhage. *J.A.M.A.*, Feb. 21, 1948, *136*, 517–521.

Ten cases are presented with illustrative cerebral arteriograms to demonstrate the usefulness of this method in the diagnosis, prognosis, and therapeutic indications for acute and subacute phases of subarachnoid hemorrhage. Thirty-five per cent diodrast has been used since 1939 without deleterious effect or untoward complications in any patient. There is no contraindication to arteriography in the very acute phases of the hemorrhage.

The notion that spontaneous subarachnoid hemorrhage is generally the result of rupture of a cerebral aneurysm, especially in the circle of Willis, is not borne out in this small series. Six cases showed focal vascular malformations, and 3 of these had histories of recurrent episodes of bleeding and recovery. Four cases showed aneurysms of which 3 were located near the bifurcations of the carotid.

Ligation of the common carotid on the side of the lesion was done in 4 patients with vascular malformations. Two others were treated by irradiation. Three of the aneurysms had common carotid ligation done, the fourth had intracranial ligation in addition. Common carotid ligation is preferred to internal carotid ligation in that it is safer, gives little danger of convulsions or hemiplegias, requires no preparatory arterial compression, and reduces circulation only 50 per cent. All patients with vascular malformations who had ligations showed no recurrence of bleeding. One case treated by irradiation alone showed recurrence. Two others were controlled by deep roentgen therapy. The authors feel that recurrent subarachnoid hemorrhages are more likely to be the result of vascular malformations, and that ruptured cerebral aneurysms are more apt to be fatal in the first or second attack.— *J. A. Campbell, M.D.*

Olsson, Olle. Subdural hematoma. *Acta radiol.*, January, 1948, *29*, 95–99.

The author presents a case of verified subdural hematoma. This was demonstrated by encephalography. By shifting the patient's head, a subdural collection of air was made to outline the mass of the hematoma. The author goes in-

to some detail to show how air gets into the subdural space; he explains it as due to absorption in the hematoma which causes a rupture of the arachnoid. The space between the arachnoid and the membrane of the hematoma becomes filled with cerebrospinal fluid, and when this fluid is withdrawn and gas injected, the fluid is replaced by gas. In this way, the characteristic picture was obtained.—*Bernard Halper, M.D.*

NECK AND CHEST

Baclesse, F., and Dulac, G. Le radiodiagnostic des tumeurs malignes du rhinopharynx. (Roentgen diagnosis of malignant tumors of the rhinopharynx.) *J. de radiol. et d'électrol.*, No. 1–2, 1948, *29*, 24–36.

Roentgen examination of the rhinopharynx constitutes an excellent means of exploring this region and one frequently neglected. This method permits not only the diagnosis of a tumor, but also determines its extension, and also provides exact localization of treatment fields to be used during irradiation.

The authors give their technique for diagnostic films of this region: lateral projection, Bowen-Hirtz (with and without Bucky), lateral projection with 40 per cent lipiodol when required, and when indicated, planigraphy.

Lymphosarcomas, epitheliomas and lymphoepithelioma which represent the most frequent malignant tumors in this region produce pathological shadows which are often similar. However, certain localizations, as in the case of epithelioma of the eustachian tube, have a special appearance which permits their differentiation from other epitheliomas on a level with the posterior superior wall (pharyngeal tonsil and fossa of Rosenmüller), i.e., the epitheliomas of the eustachian tube invade the bones of the base of the skull. Films of the base of the skull (Bowen-Hirtz) are indispensable in examination for cancer of the rhinopharynx. Lymphosarcomas do not invade the bones and since their extension is not always accompanied by pain or signs of paralysis from involvement of the nerves of base of skull it is clear that roentgenography is a prime aid in diagnosis and prognosis.

The article contains many excellent roentgenograms of (1) normal rhinopharynx, (2) tumors of the rhinopharynx.—*William M. Loehr, M.D.*

Clagett, O. Theron, and Schmidt, Herbert W. Surgical management of acquired stricture of the esophagus with esophagobronchial fistula and bronchiectasis of entire right lung; report of a case. *Surgery*, February, 1948, *23*, 221–226.

The authors report the case of a man twenty-five years of age, who had swallowed lye at the age of two years. Periodic esophageal dilatations had been performed for the resulting stricture. When he was twelve years of age he had pneumonia after which bronchiectasis developed. Roentgenograms of the thorax revealed an extensive suppurative process that involved the entire right lung. Bronchographic study demonstrated diffuse saccular bronchiectasis of the right lung. There was a small area of cylindric bronchiectasis in the base of the left lung. The lower portion of the esophagus was examined with the aid of barium which was injected into the stomach through the gastric stoma and a fistula from the esophagus to the right bronchus was demonstrated. A silk thread was passed through the strictured area so that dilatations could be performed. The strictured esophagus was re-utilized as a tube to convey food. Pneumonectomy was performed on the right lung and the patient had an uneventful recovery.—*Frank H. Marek, M.D.*

Burnett, W. Emory, and Caswell, H. Taylor. Lobectomy for pulmonary cysts in a fifteen-day old infant with recovery. *Surgery*, January, 1948, *23*, 84–91.

Fischer reported a case of a large thin-walled pulmonary cyst with bronchiolar mucosa and musculature. The cyst involved the right upper and middle lobes. Follow-up of the patient showed normal growth and development after lobectomy. Gross reported a similar case, that of a twenty-three day old infant, in which pneumonectomy was done. The patient in the case reported by the authors was fifteen days old, which shows the feasibility of major pulmonary operations on infants suffering with congenital pulmonary air cysts. This type of cyst is distinguished from diaphragmatic hernia or eventration of the diaphragm by the demonstration of viscera in either normal or abnormal position. A single, large cyst must be distinguished from pneumothorax. Infection may confuse the picture, as in the case reported by Rosemond and Caswell in which there was

pressure from pyopneumothorax in the first month of life. The remarkable ease with which lobectomy can be done on infants was noted in this patient and was described by Fischer and co-workers and by Gross. The hilum forms a small pedicle which makes hilar ligation simple and easy and which shortens the operation greatly.—*Frank H. Marek, M.D.*

Lame, Edwin L., and Pendergrass, Eugene P. Calcification of pleura and lung. *Radiology*, June, 1947, *48*, 636–641.

A case report of extensive calcification in left thorax studied by body section roentgenography with theoretical discussion as to the site of the calcification. The calcification might be largely lung or largely pleura. Patient is still living.—*E. C. Baker, M.D.*

Holm, Mogens, Holm, Sigrid, and Winge, Knud. Results and experiences from mass examinations for tuberculosis in Copenhagen 1946. *Acta tuberc. Scandinav.*, 1948, *22*, 97–124.

In the last decade fluorophotography has been widely adopted as a means of detecting tuberculosis. It was not until 1935 that a complete report was given before a German Congress of Roentgenologists. They stress four ideal points as to what we wish to learn from reported statistics: (1) How many cases are discovered that otherwise would not have been discovered until latent symptoms developed? (2) How many of those discovered are suffering from open tuberculosis? (3) How many are in need of medical treatment and how many might recover without treatment? (4) How are the figures obtained under 1–3 with regard to age and sex? The British, Australian and American statistics are based on roentgenography and eventually clinical examination of suspects. The latter statistics varied from 0.10 to 0.5 per cent active cases and 0.09 to 0.29 per cent open cases. Examinations in the Copenhagen Central Dispensary over a period from 1935 to 1945 revealed 2.4 per cent active tuberculosis. Examinations of households with new cases of open tuberculosis revealed 3.7 per cent cases of disease. They stress the close cooperation of all physicians in constantly referring all suspected persons for roentgen examination.—*Robert J. Reeves, M.D.*

Rosenthal, Sol R., Leslie, E. I., and Loewinsohn, E. BCG vaccination in all age groups: methods and results of a strictly controlled study. *J.A.M.A.*, Jan. 10, 1948, *136*, 73–79.

This accumulation of facts and figures represents the longest continuous experiment on BCG vaccination in the United States (thirteen years). Six groups are being followed and this present paper presents the results in two of them: newborn infants delivered at Cook County Hospital of Chicago, Illinois (study in progress ten years), and infants born of tuberculous parents anywhere in Chicago, Illinois (study in progress seven years).

Among 2,831 newborn infants, not in household contact with tuberculosis, there were 11 cases of tuberculosis with 1 death in the vaccinated group and 39 cases of tuberculosis with 7 deaths in the control group.

Among 256 newborn infants, when tuberculosis was present in the household and when isolation in foster homes was practiced in the controls and vaccinated alike, there were 2 cases of tuberculosis with no deaths in the vaccinated groups, against 5 cases with 4 deaths in control group.

A scarification method of multiple punctures is used instead of the oral method or intracutaneous route of injection. As a result, no local unfavorable sequelae followed.—*M. M. Manalan, M.D.*

Chatton, P., and Maleki, A. Le tomogramme thoracique de profil chez le sujet normal. (The lateral thoracic tomogram in the normal subject.) *J. de radiol. et d'électrol.*, No. 1–2, 1948, *29*, 51–54.

These workers in the Department of Radiology at the St. Charles Clinic, Montpellier, under the direction of Prof. Paul Lamarque, discuss very concisely yet completely the planigraphic appearance of the normal structures seen in lateral sections through the thorax. Skeletal, mediastinal, and bronchovascular structures are discussed. Particularly useful is the section cut 3 or 4 cm. to the right of the median line in which is contained the majority of the bronchovascular and intrahilar elements.

Exact morphological study of lesions poorly seen en face, localization of all lesions in respect to pulmonary zones, measurements of diameter

of aortic arch, pulmonary artery, and descending aorta are possible with these planigrams.—*William M. Loehr, M.D.*

ABDOMEN

Bockus, H. L. Recent advances in treatment in field of gastroenterology. *J.A.M.A.*, Jan. 31, 1948, *136*, 293–299.

A large number of patients presenting themselves to the internist and to the general practitioner for relief of gastrointestinal complaints have no recognizable organic disease in the alimentary tract or elsewhere. Although proof is lacking to support the psychogenic origin of common gastrointestinal disorders like peptic ulcer, regional enteritis and ulcerative colitis, a careful psychologic survey on many of these patients is highly suggestive of the presence of an important psychogenic component in their illness.

Approximately 2,000 persons die in the United States every year of carcinoma of the esophagus. In 35 to 50 per cent of cases, the lesion is located in the lower end of the esophagus.

Approximately 10 per cent of carcinomas of the stomach occur in the cardiac end. By means of the transthoracic approach a number of surgeons are now resecting the lower part of the esophagus or the lower part of the esophagus and the upper part of the stomach and reestablishing continuity by doing an esophagogastrostomy in the treatment of these lesions. The mortality in the large group of patients operated on by Sweet is now somewhere between 12 and 15 per cent. It now seems obvious that some patients with carcinoma of the lower part of the esophagus and of the cardiac end of the stomach may be cured by radical transthoracic resection. It is quite possible that the five year cure rate may be as high as or higher than that encountered in radical resection for carcinoma at the lower end of the stomach.

Levy and Siler suggested that amino acid mixtures might prove of benefit in the management of peptic ulcer. A perusal of recent publications dealing with amino acid mixtures as the keystone for treatment of active peptic ulcer supplies no convincing evidence of their superiority over milk formulas in the well nourished patient. There is every reason to believe that reliance on these mixtures alone and ignoring of the many items utilized by the experienced gastroenterologist in all-out therapy is unfortunate.

Two hormones elaborated in the body, enterogastrone, a chalone released by contact of the intestinal mucosa with fat, and urogastrone, recovered from urine, have been used by various workers in patients with chronic peptic ulcer. Some success in preventing recurrence of symptoms was obtained, but Bockus says that up to this time no evidence has been forthcoming to indicate that hormones will prove of great value in the healing of ulcer. If they are to have any value in ulcer disease, evidently it will be in the prevention of recurrence.

In approximately 10 to 15 per cent the results of medical treatment are unsatisfactory because of obvious complications or intractability. It is only in this latter group that surgical procedures of any kind are considered. In a large number of these because of obstruction or plastering of the ulcer, vagotomy alone should not be undertaken. By these criteria, section of the vagus fibers might come up for consideration in approximately 5 per cent of ulcer patients not operated on. Even though vagus section accomplished the results expected of it, it should never be recommended until adequate medical therapy, including attention to all known etiologic factors, has been given a thorough trial.

Bockus believes that the operation of complete intrathoracic vagus interruption or resection remains an experimental procedure. A complete interruption of the vagus innervation of the stomach is not always possible, and it may yet prove to be undesirable because of late side effects. Only patients with duodenal ulcer who have a strongly positive reaction to the insulin test and a history suggesting emotional influences in the initiation of attacks and who have not responded to really adequate medical treatment should be considered as candidates for the experiment. Gastric ulcer or delay in stomach emptying in duodenal ulcer should be looked on as contraindications. At this time intrathoracic vagotomy can be considered the operation of choice only in those patients who have experienced recurrence of ulcer after a large subtotal gastrectomy which has included ablation of the antral and pyloric mucosa.—*Samuel G. Henderson, M.D.*

Feldman, Maurice. Mucosal deformities of the greater curvature of the stomach. *Radiology*, August, 1947, *49*, 152–161.

Irregularities of the greater curvature side of the stomach due to abnormal spacing and other deformities of hypertrophied mucosal folds are not infrequent. Marked thickening of the folds is usually not present in carcinoma but is noted in Hodgkin's disease, lymphoblastoma and syphilis. In some cases an exact diagnosis cannot be made by roentgen methods alone. Repeated examinations, clinical investigation, gastroscopic studies and clinical follow-ups are essential for accurate diagnosis. The author presents a group of cases demonstrating greater curvature irregularities which simulated primary gastric lesions.—*J. N. Ané, M.D.*

Benson, Clifford D., and Penberthy, Grover C. Congenital duodenal obstruction (intrinsic obstruction). *Arch. Surg.*, January, 1948, *56*, 58–65.

Congenital atresia and stenosis of the small intestine has in the past carried a high mortality. The authors state that approximately 30 operative recoveries are recorded. It is said to occur only in about 1 out of 20,000 cases.

Early recognition and operative relief of complete obstruction is the keynote to success in handling these cases. Most cases begin vomiting on the first day after birth, after being fed. Peristaltic waves can frequently be seen in the epigastric region, some of which progress downward and to the right. If the obstruction is below or at the ampulla of Vater the vomitus will be bile stained. Roentgenograms of the abdomen in the upright and inverted position will usually show air in the stomach and duodenum, possibly both. Failure to show gas in the distal small intestine and in the large intestine is of some help in localizing the lesion.

Infants with partial or complete obstruction, at or immediately distal to the ampulla of Vater, must have a short circuiting procedure done without delay. Dehydration should be corrected first. The surgical procedure of choice for lesions of this type are gastroenterostomy and duodenojejunostomy, the procedure of choice depending upon the case. Most surgeons that have had experience with lesions of this type prefer the duodenojejunostomy. This is because the neutralization of the stomach acid and consequent loss of appetite, as well as poor drainage of the duodenal loop, often follow the gastroenterostomy. Gastroenterostomy is at times preferred because of the technical difficulty of anastomosing a large distended duodenum to a small jejunum that has never functioned.

The authors present 2 cases of atresia. One was operated on the fourth day following birth and the beginning of symptoms. A gastroenterostomy was done. Vomiting recurred during the second postoperative week. This was believed to be due to a poorly functioning stoma, but the infant's condition would not permit more surgery. The patient died of pneumonia one month after operation. The autopsy showed a gastroenterostomy stoma which was contracted and which was apparently functioning inadequately. The second case, after adequate preoperative preparation, was brought to surgery and a gastroenterostomy performed. She was seen five months later and was developing normally.

One case of partial stenosis was presented with attention being called to this being a nonemergency procedure. This case had vomited intermittently since birth. His inability to retain solid foods caused his diet, of necessity, to be entirely liquid. Nine months after a gastroduodenostomy a regular diet was being tolerated without difficulty and the patient was well. —*Robert J. Reeves, M. D.*.

Weig, Clayton G. Benign ulceration within a duodenal diverticulum. *Radiology*, February, 1947, *48*, 143–147.

The author reports a case of benign ulceration within a duodenal diverticulum and discusses the pathogenesis of the lesion, as well as discussing the palpation of the patient in the horizontal position as a vital part of the roentgen examination. The clinical source of bleeding in the gastrointestinal tract of this particular lesion is also discussed in the text of the case report. The rarity of the lesion is stressed by the author.—*Moris Horwitz, M.D.*

Koenig, E. C., and Culver, Gordon J. Retroperitoneal perforation of the duodenum. *Radiology*, February, 1947, *48*, 164–167.

The authors report a third case of retroperitoneal perforation of the duodenum, having previously reported 2 cases in December, 1944 (*Radiology*, Vol. 43, pp. 563–571). The first 2 cases reported by the authors were due to blunt trauma to the back, while the third was due to an ulcer of the posterior wall of the duodenum. The appearance of the retroperitoneal distribu-

tion of gas following perforation is stressed.—*Moris Horwitz, M.D.*

Evert, John A., Black, B. Marden, and Dockerty, Malcolm B. Primary nonspecific ulcers of the small intestine. *Surgery*, February, 1948, *23*, 185–200.

The authors report a review of cases in the literature and 14 cases that were encountered at the Mayo Clinic with a total of 45 cases since 1931. Roentgenologic demonstration of a small intestinal ulcer is rarely possible. However, on 2 occasions constricting ulcers have been demonstrated by means of a barium meal. This condition should be thought of in the following groups of patients: (1) patients who present a problem of gastrointestinal hemorrhage, acute or chronic, but roentgenologic evidence of abnormality of the esophagus, stomach, duodenum or colon is not apparent; (2) patients who have symptoms and signs of peritonitis suggestive of a ruptured peptic ulcer, but laparotomy does not reveal gastric or duodenal perforation, and (3) patients who present a problem of recurrent postprandial pain suggestive of peptic ulcer, but roentgenological evidence of gastric or duodenal ulceration is not present.

Nonspecific localized ulcerations of the jejunum and ileum are so similar pathologically as to justify their classification as a group under the name "primary" or "simple" ulcers. The etiology of primary ulcers is unknown. The symptoms of primary ulcer for the most part are secondary to complications of perforation, bleeding or obstruction. The mortality rate in patients who suffer from primary ulcer is high. The lesion has been recognized during life only after some complication has led to surgical intervention.—*Frank H. Marek, M.D.*

Sampson, D. Alan, and Stauffer, Herbert M. Rupture of small intestine complicating injury of pelvis. *Radiology*, July, 1947, *49*, 80–85.

Traumatic rupture of the small intestine is frequently unattended by even the slightest contusion of the superficial tissues. It is often rapidly fatal.

Usually there will be signs of rapidly developing peritonitis and pneumoperitoneum, but in some instances the diagnosis may be obscured. The peritonitis may be localized in the pelvis. Vomiting after recovery from shock is a valuable early symptom. Rigidity is the most valuable single sign. Suspicion may depend only upon a rise in pulse rate or the absence of peristaltic sounds. Suspected cases should be subjected to roentgen examination for generalized or localized pneumoperitoneum. In the presence of other severe injury, the possibility of intestinal rupture may be overlooked.

With the exception of two cases in which recovery followed drainage of walled-off abscesses, the authors found no record in the literature of survival when operation had been delayed beyond a number of hours. Therefore they report 2 cases which are remarkable in that the rupture was unsuspected for several months, and which were restored to normal function by operation.

Case 1, male aged forty-one. Roentgen studies of the gastrointestinal tract were carried out two months after severe pelvic injury, because of vomiting and persistent abdominal pain. Films revealed narrowing of a segment of small intestine in the lower central abdomen with loss of normal mucosal pattern and partial obstruction. Laparotomy disclosed complete transverse laceration of the terminal ileum, which had been walled off by inflammatory tissue. End-to-end enterostomy was performed.

Case 2, female aged thirty. Gastrointestinal studies were done one year after injury, because of intermittent right lower quadrant pain. An attenuated segment of terminal ileum and marked distortion of the cecum were demonstrated. Laparotomy revealed the transverse colon and a loop of ileum forming an inflammatory mass, within which was found a perforation of the ileum. The opening was closed and an ileocolostomy performed.—*Arthur A. Brewer, M.D.*

Fishback, H. R., Jr. Carcinoma of the transverse colon in a fifteen-year old boy. *Radiology*, February, 1947, *48*, 168–171.

The author reports a case of transverse colon carcinoma in a fifteen year old boy, in addition to giving a short summary of previously reported cases in the literature. The author stresses the fact that more cases of early carcinoma in children and adolescents will probably be diagnosed if this particular pathologic entity is kept in mind during the roentgen examination. He also points out the fact that there is a slightly higher incidence of malignant lesions of the gastrointestinal tract in the cecum in

youth as compared to adults and also states that the lesions are apparently more malignant in the left half of the colon than in the right half of the colon. In his review of the literature, he has noted that of 38 young patients with carcinoma of the colon only 1 survived a period of seven months following discovery of the lesion. The author's case was still alive and well nine months after surgical extirpation.—*Moris Horwitz, M.D.*

FELSON, BENJAMIN, and BERNHARD, C. MELVIN. Roentgenological diagnosis of appendiceal calculi. *Radiology*, August, 1947, *49*, 178–191.

Appendiceal calculi, as discussed in this paper, should be distinguished from the non-calcified foreign bodies in the appendix such as fecaliths.

This study is based on 100 cases reported in the literature and 10 cases from the authors' personal experience with detailed histories and roentgenographic findings in the 10 cases.

The importance in preoperative diagnosis is apparent, as the authors state that practically all appendices with calculi sooner or later will result in acute appendicitis and that approximately 50 per cent will perforate. Once a diagnosis can be made surgery is to be recommended whether the patient has any clinical symptoms or not.

Roentgen diagnosis is made by the finding of laminated calcification in the region of the appendix. Diagnosis can be confirmed in most instances by fluoroscopy at the time of barium enema.—*E. A. Addington, M.D.*

THOMAS, SYDNEY F. Appendiceal copraliths; their surgical importance. *Radiology*, July, 1947, *49*, 39–49.

Appendicitis is still a major problem. Roentgen studies help in complications and in demonstration of copraliths containing calcium. With such demonstration surgery is indicated. Copraliths may be laminated and must be differentiated from gallstones and ureteral calculi and lymph node calcifications. Copraliths occur frequently in appendiceal perforation. Appendiceal fecaliths are radiopaque in possibly 25 per cent of cases. Six case reports are given.—*E. C. Baker, M.D.*

MAGEE, R. K. Recto-urethral fistula. *Lancet*, January 24, 1948, *1*, 140.

Report of one case in a thirty-three year old male German prisoner of war. The fistula was demonstrated by filling the bladder with radiopaque dye and a roentgenogram taken while the patient was straining to empty his bladder. The fistula was so large that a cystoscope passed easily from the urethra to the anus. The appearance, size and position of the fistula was most suggestive of a congenital connection between the urinary and intestinal tracts.—*J. S. Summers, M.D.*

SELTZ, HERMAN. Adrenal tumor of the liver in a child. *Radiology*, 1947, *49*, 86–89.

Adrenal cell rests have been described throughout the upper abdomen. Such accessory adrenal tissue may undergo malignant transformation. Adrenal tumor arising in the liver is rare; the author reports a case of aberrant adrenal tumor of the liver in a nine year old boy.

The patient presented a large, discrete, firm, non-tender mass in the epigastrium and left hypochondrium. Gastrointestinal studies revealed displacement of the body of the stomach to the left and posteriorly, the pyloric portion and duodenal bulb cephalad, the descending duodenum markedly to the right, and the jejunum and transverse colon inferiorly. The marked widening of the duodenal loop suggested a lesion in the region of the head of the pancreas, but the posterior displacement of the stomach was inconsistent with a retroperitoneal tumor; therefore the mass was considered to be in the region of the left lobe of the liver.

Laparotomy revealed a large, gray, cyst-like mass about the size of a grapefruit protruding from the inferior aspect of the left lobe of the liver. Satellite discrete nodules were seen scattered throughout the remaining portion of the left lobe of the liver. Two small nodules and aspiration material from the cyst-like mass were removed for histopathologic study. The pathological diagnosis was adrenal tumor in the liver.

At time of the report nine months later, there had been no change in the general condition nor in the size of the tumor.—*Arthur A. Brewer, M.D.*

DASHIELL, GRAYSON F., and PALMER, WALTER L. Carcinoma of the pancreas; diagnostic criteria. *Arch. Int. Med.*, February, 1948, *81*, 173–183.

In this paper 90 cases are reviewed and com-

pared with those in other studies. The anatomic location of the lesion in this series is discussed and as would be expected the head of the pancreas was usually involved in patients with jaundice. Metastases were most frequent to the liver and the regional lymph nodes. The average age of the patients on their admission to the hospital was fifty-five years and the sex ratio was 2 males to 1 female. By far the predominant chief complaint was pain, followed in frequency by jaundice and loss of weight. Pain is extremely important as an early symptom and although variable in character, it was typically dull and persistent and located high in the abdomen.

Difficulty is encountered in regard to the patients without jaundice, who comprise one-third of all those affected. Diarrhea is less frequent, but nevertheless is an extremely important symptom, but it tends to be persistent, and not explained in the usual ways and not relieved by the usual measures. Steatorrhea is even more significant and when coupled with the other symptoms mentioned is almost pathognomonic.

Notwithstanding the lack of a radiopaque dye which can be selectively absorbed by the pancreas, the indirect evidence afforded by careful roentgenographic examination greatly enhances the chance of correct diagnosis. By excluding lesions such as carcinoma of the stomach, the study may provide evidence of an extrinsic mass in the region of the pancreas. In this series roentgenological evidence suggestive of the lesion was present in about one-half of the patients so examined. The signs most commonly noted were irregularities in the duodenal contour, distortion and displacement of the stomach, deformities of the duodenal bulb and in 4 cases expansion of the duodenal loop.—*Eugene J. McDonald, M.D.*

Tocantins, Leandro M. The hemorrhagic tendency in congestive splenomegaly (Banti's syndrome); its mechanism and management. *J.A.M.A.*, Feb. 28, 1948, *136*, 616–621.

The article is based on observations made on 22 patients for whom a diagnosis of Banti's syndrome was indicated and who had had bleeding in one form or another. It is felt that the cause of excessive bleeding is due to multiple defects in hemostatic function: dilatation of veins which are branches of the portal system, thinning or ulceration of the mucosa overlying these dilated veins, venous hypertension, thrombopenia and hypoprothrombinemia.

The phase of acute bleeding is managed by judicious replacement of blood and fluids so that there is enough blood to maintain circulation without attempting to raise the hemoglobin concentration to normal levels. Vitamin K is employed during the hemorrhagic phase. If these conservative measures fail, a small intestine tube with a distensible bag is introduced into the stomach and pressure exerted against the cardia for several hours. The stomach can be aspirated during this time to determine when bleeding has stopped.

The results from splenectomy are more gratifying when the cause of splenomegaly is thrombosis of the splenic vein. But even if the cause is an intrahepatic block, the removal of the spleen cuts out about 40 per cent of the blood load into the portal vein and aids in that manner. Following splenectomy, if hematemesis continues, the varicosities should be injected with a sclerosing solution via an esophagoscope. The thrombopenia is often corrected by splenectomy. The hypoprothombinemia can be overcome, in those patients whose hepatic function is not too far deranged, by the use of vitamin K and supplementary diet designed to aid hepatic function. The operative mortality is about 25 per cent. Half of these are due to hemorrhage from the operative site, and the remainder are due to mesenteric or portal thrombosis and pulmonary embolism.—*M. Manalan, M.D.*

GENITOURINARY SYSTEM

Scholl, A. J. Peripelvic lymphatic cysts of the kidney. *J.A.M.A.*, Jan. 3, 1948, *136*, 4–7.

Peripelvic cysts of the kidney are rare, usually small, clinically insignificant cysts in the hilum. They are believed to represent lymphatic ectasis, either congenital in origin or secondary to an inflammatory disease with resultant obstruction of the lymphatic trunks at the hilum of the kidney.

Although these cysts are usually small and found only incidentally at autopsy, they may become very large and multilocular, containing large amounts of fluid and even calcareous material. By their size, they cause considerable distortion of the kidney with compression of the pelvis and structures of the hilum of the kidney.

The author discusses 2 cases, both of which he operated on for large peripelvic lymphatic cysts. These cases were of interest because of

the origin and large size of the cysts, their apparent relationship to hypertension, and the similarity of the diagnostic signs to those of renal tumors.

Surgical removal of the cystic kidney in each case was followed by considerable alleviation of the hypertension.—*W. Gallo, M.D.*

SKELETAL SYSTEM

Greenfield, Henry. Chronic salmonella bone infection. *Radiology*, June, 1947, *48*, 633–635.

Systemic infection with organisms of the *Salmonella* group is not of rare occurrence. Most cases are of an acute toxic type, with severe constitutional reactions, mainly in infants and children. There are only a few reported cases with localized chronic pyogenic manifestations.

The case is reported in a male, aged twenty, who injured his ankle in 1940 and had recurrent bouts of pain and swelling. Roentgen studies, in 1942, revealed a cystic degenerative process, involving the distal metaphyseal portion of the tibia and extending into the medial malleolus. In 1943, cellulitis developed, subsiding several weeks after incision and drainage. In 1945, pain and swelling recurred, and roentgen studies again showed cystic changes, with some condensation of the walls of the larger cysts. There was no expansion of the shaft, and the cortex was not involved. The ankle joint appeared normal.

The lesion was explored surgically. Surgically and histopathologically, the diagnosis was chronic medullary bone abscess. Cultures revealed *Salmonella Oranienburg*, which is one of the paratyphoid group of organisms. The members of this group resemble each other closely, being gram-negative motile rods. Differentiation by morphological cultural and sometimes even sugar fermentation methods is impossible. Agglutination by specific antisera and agglutination absorption are required for final differentiation.—*T. J. Wachowski, M.D.*

Heilbrun, Norman, and Kuhn, William G., Jr. Erosive bone lesions and soft-tissue ossifications associated with spinal cord injuries (paraplegia). *Radiology*, June, 1947, *48*, 579–593.

A series of 99 observed paraplegic cases was divided into four groups depending upon the severity of the particular erosive bone change. These groups were tabulated according to the interval between injury and roentgen survey. The extent of the erosive change in most cases was directly related to the severity, size and duration of the decubitus sores. Eighty-five per cent of this series had sores over the sacrum, the trochanters or the ischial tuberosities.

The morphological characters of numerous varieties of soft tissue ossifications are described as observed by the authors. The pathological manifestations of the erosive lesions, the sequence of these changes as reflected through the roentgen findings and a discourse on the probable mechanisms involved, are presented.

Pathologically, devitalization of the soft tissues over bony prominences eventuate in erosive lesions, become evidenced by an infection overlying the bone, extend to the superficial layers of the bone and lead to resorption of the cortex and underlying trabeculae.

Various aspects of trauma and nervous influences as etiological agents are discussed in considerable detail. The authors state that although soft tissue trauma may play an important role in the etiology of soft tissue ossifications, the neurogenic influence cannot be disregarded.—*E. D. Hudack, M.D.*

Klein, A., Joplin, R. J., and Reidy, J. A. Treatment of slipped capital femoral epiphysis. *J.A.M.A.*, Feb. 14, 1948, *136*, 445–451.

Patients with pronounced epiphysiolysis are treated by arthrotomy, reposition of the displaced epiphysis on the neck and fixation by means of a three flanged nail. Patients with only minimal slipping are treated by lateral nailing in situ, without arthrotomy and without correction of the early deformity.

Patients suspected of a slipped epiphysis should immediately be forbidden any weight bearing on the affected limb. Diagnosis is established from anteroposterior and lateral roentgenograms. A line is drawn along the superior border of the femoral neck in the lateral view and extended across the epiphyseal line. A second line at right angles is drawn from the first line to the superior edge of the displaced epiphysis. If this distance is 1 cm. or more, the slip is considered pronounced; if less than 1 cm., it is minimal and necessitates no alteration of position. Similar roentgen views are taken during the operative procedure to insure proper reduction, and the lateral view is considered most essential.

Nailing insures against further slipping and

accelerates fusion of the epiphyseal line. Early postoperative mobilization is fostered by Buck's extension for cases of nailing in situ and balanced traction for cases of open reduction. Mobilization is further promoted by encouraging walking with the aid of crutches as early as two weeks after operation. Manipulation is permissible only in the recent acute slip. Traumatic arthritis follows inadequate replacement of the epiphysis. Aseptic necrosis was not manifest in any case. To correct a pronounced slip, osteotomy at the epiphyseal plate gives a better result than one performed at any other site.

Fifty-one cases in 45 patients are reported. Fifteen per cent showed bilateral slipping. Approximately 60 per cent were minimal and 40 per cent were pronounced. With hips nailed in situ one can expect 90 per cent normal motion and 96 per cent normal hip function. Those nailed after arthrotomy will give 85 per cent normal motion and 92 per cent normal hip function. These figures obtain for observation periods of three years, but are better with longer follow-up.—*J. A. Campbell, M.D.*

Lewis, Raymond W. Roentgen diagnosis of pigmented villonodular synovitis and synovial sarcoma of the knee joint. *Radiology*, July, 1947, *49*, 26–38.

This term embraces a variety of previous terms. The condition may be single or diffuse. The single lesions are not diagnostic. The diffuse type is diagnostic and shows normal, young adult bones with excessive synovitis, often nodular. History is long, disability minor. Sarcoma may be suspected if nodular masses extend beyond capsule, contain lime, or invade bone.

Surgery does not relieve villonodular synovitis. Exploratory biopsy should be done. Case reports are given. The illustrations are excellent.—*E. C. Baker, M.D.*

Vaughan, C. E., and Stapleton, J. G. Osteochondritis dissecans of the ankle. *Radiology*, July, 1947, *49*, 72–79.

Osteochondritis dissecans is an aseptic necrosis of subchondral bone and the overlying cartilage. Usually bone adjacent to an articular surface is affected. The fragment may separate and form an intra-articular loose body. Recurrent pain, effusion, weakness, and osteoarthritic changes constitute the usual sequence of events if the condition is untreated.

Most writers refer to involvement of the knee joint, but lesions involving the hip, elbow, ankle, and metacarpophalangeal joints have been reported.

Osteochondritis of the ankle is rather uncommon. The authors found 20 cases reported up to 1941, to which they add 4 cases.

Trauma is the usual etiological factor and was the major cause in their cases. Characteristic roentgen findings are similar to those found in other joints. The lesion is best seen in an oblique view. A small button-like fragment of devitalized bone is visualized lying in a depression at the upper margin of the trochlea of the talus on either the medial or lateral side. Usually it is demarcated from the adjacent bone by a radiolucent line. In the authors' 4 cases the condition was not bilateral and no loose bodies were formed, but both eventualities do occur.

Details of 4 proved cases are reported. Treatment consisted of operative removal of the fragment and curettage of the necrotic bone. The immediate results in all cases were satisfactory and the patients were walking without discomfort when discharged.—*Arthur A. Brewer, M.D.*

Rosenberg, F. Edward, and Arens, Robert A. Gout; clinical, pathological and roentgenographic observations. *Radiology*, August, 1947, *49*, 169–177.

The authors stress the importance of the fact that gout progresses in an orderly and developing pattern of (a) larval period, (b) a period of acute articular attacks, and (c) a period of chronic articular gout. The exact nature of the anomalous uric acid metabolism in this disease is not known. Histopathological features of the disease are fundamentally similar regardless of site of involvement.

The roentgenological appearance of gout depends on the state of advancement of the pathological changes. The earliest changes are not expected to be seen roentgenologically until late in the second period and should be looked for in the bunion joint. The first notable change is the appearance of a zone of osteoporosis on the medial aspect of the base of the first metatarsal and first phalanx. As the disease progresses the zone of osteoporosis becomes cystic. In the next stage the joint space is narrowed as a result of erosion and by gouty pannus. The continued use of such joints produces traumatic changes as a result of which marginal hypertrophic

changes also occur. Extensive destructive changes may be seen where tophaceous deposits in ends of bones expand greatly. Entire joints may disappear at such sites leaving bony stumps projecting into a formless tophaceous mass. An occasional subchondral expanding bone lesion of gout can stimulate certain bone tumors, e.g., giant cell tumor or osteogenic sarcoma. Tophi have been referred to as "chalk stones" because of the white, chalky appearance of their contents. This material does not contain notable quantities of calcium and consequently tophi are not particularly radiopaque.

This is an excellent orderly exposition of gout. —*E. A. Addington, M.D.*

Paul, Lester W., and Moir, William W. Roentgen diagnostic aspects of chronic arthritis and bursitis. *Radiology*, July, 1947, *49*, 6–17.

Clinically chronic arthritis may be generalized, single, or limited to the periarticular tissues. Roentgenograms do not always identify the type. Early diagnosis is helpful in degenerative arthritis. Rheumatoid arthritis starts in the hand and progresses to the trunk. Roentgen features are periarticular swelling, decalcification and narrowing of joint space. Hypertrophic changes may appear late. Rheumatoid arthritis of the spine (Marie-Strümpell) shows earliest change in sacroiliac joints. It progresses through the spine and usually shows ligamentous calcification.

Degenerative joint disease shows marginal spurring, thinning of discs and facet joint changes. Malum coxae senilis is included in this type. Roentgen characteristics vary in weight-bearing and non-weight-bearing joints. In hip and knee there is asymmetrical narrowing. Spurs may appear and also increased density of bone margins. In the spine, spurs are the usual manifestation. These spurs with thinning of discs may narrow spinal foramina.

Periarticular disease is represented by calcific deposits in the soft tissues of the shoulder.—*E. C. Baker, M.D.*

Angevine, D. Murray. The pathology of rheumatic disease. *Radiology*, July, 1947, *49*, 1–5.

The etiology is usually unknown. The pathological process involves the synovial membrane and/or the articular cartilage. The classification followed is that of the American Rheumatism Association—acute infections of known etiology, chronic infections of known etiology, probably infectious, and degenerative joint disease or osteoarthritis. Acute infections of known bacteria follow when such bacteria are carried by the blood to the synovial membrane. The anatomical structure of the synovial villus is believed to be a factor in localization of joint infections. Tuberculosis is the important etiological agent of known chronic infections.

Under "probably infectious," rheumatic fever, rheumatoid arthritis, Marie-Strümpell spondylitis and Still's disease are briefly described. Rheumatic fever is transient. Rheumatoid arthritis frequently starts in the hands. The fusiform swelling is caused by edema. With progression of the lesion there is infiltration with various white blood cells and thickening of the arteries. The formation of a pannus is the next change. Marie-Strümpell spondylitis usually affects young male adults. It involves the synovial membrane of the costovertebral articulations and progresses to ankylosis. Still's disease is found in children. The lymph tissues are enlarged. Deformities are common. Histopathology shows chronic inflammation.

Degenerative joint disease is a more desirable term than osteoarthritis. There is degeneration of cartilage which frays and may be lost with change in the underlying bone.

In acute and chronic bursitis and tenosynovitis the synovial membrane is involved with infiltration of cells and edema. Calcium deposits may occur.—*E. C. Baker, M.D.*

Dell, J. Maxey, Jr. An unusual roentgen shadow in chloroma. *Radiology*, January, 1947, *48*, 61–62.

This is a case report of an extensive chloroma involving the sternum, mediastinal lymph nodes and encasing the thoracic spine. The patient had monocytic leukemia. There was an agreement between the roentgen and postmortem findings as to the extent and location of the chloroma.—*J. H. Harris, M.D.*

Saenger, Eugene L. Unilateral paraspinal abscess. *Radiology*, March, 1947, *48*, 256–259.

The author reports what he believes is the only recorded case of unilateral paraspinal abscess. The disease was due to tuberculosis at T-5 and the abscess protruded to the left extending along the entire left side of the

thoracic spine. The patient was a fifty-seven year old white male.

The normal linear thoracic paraspinal shadow or the posteromesial pleural line is formed by the mediastinal pleural border of the left lung; due to the presence of the azygos vein, the line is usually not seen on the right. The line is seen only occasionally in infants and children, it is frequently seen in adults, and is most often visualized in the older age groups.

The globular or fusiform abscesses are almost pathognomonic of tuberculosis, but the normal paravertebral shadows can also be displaced bilaterally by extension of a vertebral body neoplasm, pyogenic vertebral osteomyelitis, vertebra plana (acute stage), osteitis deformans, and vertebral osteochondritis. Paravertebral abscess may be simulated by rupture of a dissecting aneurysm, by localized pleural thickening, or by localized empyema.—*Samuel H. Fisher, M.D.*

Bailey, Wilbur. Observations on the etiology and frequency of spondylolisthesis and its precursors. *Radiology*, February, 1947, *48*, 107–112.

This important fundamental presentation reviews various factors concerning the etiology of spondylolisthesis. The author is particularly concerned with the genesis and frequency of vertebral isthmus defects.

Discussing the evidence in favor of trauma as a cause for the defect seen in spondylolisthesis, the author calls attention to the experimental evidence which shows that fractures in the neural arch in the lower lumbar region in fetuses and stillborn children may easily be produced by hyperflexion of the spine even though of only moderate degree and produced with little force. In favor of the traumatic theory also is the fact that neural arch defects of the type seen in spondylolisthesis have not been described in fetal skeletons or newborn babies in the many hundreds of examinations in which they were sought.

When the genesis of such defects is considered of congenital origin, one finds much more supportive evidence. Among this evidence, one may include: (1) the fact that the neural arch defect may involve more than one segment; (2) the experience that about 25 per cent of the defects are unilateral; (3) the equal frequency with which these defects occur in different races; (4) the recent reports of spondylolisthesis in children; (5) the apparent hereditary transmission of this neural arch defect, and (6) the theoretical consideration that although normally a separate single ossification center is present through the lateral vertebral arch, it is possible that on occasion, two bone nuclei may be laid down in this region with subsequent failure of fusion similar to that seen in bipartite patellae.

The author examined the lumbosacral region in 2,080 individuals selected in the Armed Forces. Spondylolisthesis or spondylolysis was found in 4.4 per cent of this series but in only 0.5 per cent was a history of low back pain elicited.

When the roentgenograms were made with the patient erect and recumbent or with full flexion and extension, some forward motion of the vertebral body could be demonstrated in 48 per cent of the patients with isthmus defects examined by the author.

The author is convinced that spondylolisthesis or its precursors are not the result of trauma except in rare instances.—*Philip J. Hodes, M.D.*

Polley, Howard F., and Slocumb, Charles H. Rheumatoid spondylitis: A study of 1,035 cases. *Ann. Int. Med.*, February, 1947, *26*, 240–249.

This series, comprising more than one thousand cases, was studied to clarify certain aspects of this disease and to attempt to answer some of the more frequently asked questions concerning this condition.

The analysis indicates that the condition is seen in men and women in about the ratio of 9 to 1. The same sex ratio holds for the larger group of patients having rheumatoid spondylitis without peripheral rheumatoid arthritis. In the smaller group in which both conditions are present, however, the ratio remains essentially the same.

The average age at the onset of symptoms in this series was 26.7 years. More than 80 per cent of the patients had their first symptoms between the ages of fifteen and thirty-five years. The torso, peripheral joints, neck and shoulder were the sites of the original complaints, in that order. Only 3.4 per cent of the cases had their onset in the neck and shoulders. Twenty-three per cent of the cases presented symptoms in the peripheral joints as initial manifestations. Hence, in the overwhelming majority, the disease followed an ascending course after originating in the pelvis and lower spine. Those

patients with initial peripheral joint manifestations showed 87 per cent polyarticular involvement with two-thirds localized to the lower extremities.

About one-fourth of the patients suffered a fulminating progressive course. The remainder of the cases had exacerbations and remissions as a characteristic feature. The average duration of symptoms prior to the authors' examination was 8.5 years.

The sedimentation rate was found to be normal in one-fifth of the cases. It seems that the sedimentation rate tends to be higher in the cases of peripheral rheumatoid arthritis.

The roentgen evidence depends to a great degree on the amount of destruction of cartilage and subchondral bone. Although the "bamboo spine" was formerly considered to be characteristic of this condition, arthritic changes in the sacroiliac and apophyseal joints usually occur much earlier. Involvement of the sacroiliac joints is typically bilateral. Unilateral sacroiliac involvement usually suggests specific infectious disease. Although rare, absence of sacroiliac changes in rheumatoid spondylitis does occur. In the spine a distinction must be made between the marginal spurs of osteoarthritis and early calcification of the spinal ligaments.

The article contains several tables and roentgenograms to illustrate the main points.—*Robert N. Byrne, M.D.*

LINGLEY, JAMES R., and ROBBINS, LAURENCE L. Fractures following electroshock therapy. *Radiology*, February, 1947, *48*, 124–128.

The study was undertaken in order to evaluate the traumatic complications incident to electroshock therapy.

Of 230 patients who received electroshock therapy, 53, or 23 per cent, sustained fractures predominantly of the vertebrae. A total of 110 fractured vertebrae were found.

The incidence of fracture was somewhat greater in males than in females and in the younger and older rather than in the middle aged group. On the basis of these studies, the authors believe that shock therapy is contraindicated in patients with bone diseases predisposing to fracture.—*Philip J. Hodes, M.D.*

BLOOD AND LYMPH SYSTEM

FEUCHTWANGER, J. L. Arteriography of the limbs; a brief study of its value in arterial injuries. *Brit. J. Radiol.*, September, 1947, *20*, 363–367.

Arteriography should be limited to those cases where clinical diagnosis is uncertain, where exact localization of the lesion is desired, or where evaluation of the collateral circulation is needed. Since thorotrast is potentially or actually dangerous, a water-soluble contrast medium, used in demonstration of the urinary tract, is used in concentrated solution. The artery proximal to the lesion is opened and a needle is used for injection after compression of the arm or leg proximal to the injection site. Two roentgenograms are made in anteroposterior and two in lateral view with a short interval of relief of the arterial compression between them. The films can be given most value by prolonging the exposure and thus permitting maximum filling. Five to 15 milliamperes are used with exposures from three to six seconds. Ten cubic centimeters of solution are injected into the leg for each position and 5 to 8 cc. for the arm.

A series of cases with illustrations shows the findings in traumatic aneurysm of the axillary artery and popliteal artery, and thrombosis of the popliteal artery. In aneurysm the arteries leading to and from the lesion are pointed at the extremities near the lesion, while in thrombosis the ends are transverse.

Spasm in an artery may be aggravated by manipulation, as in preparation for the injection or by the actual injection itself. This may be temporary, or it may be followed by thrombosis and gangrene.—*E. F. Lang, M.D.*

ROENTGEN AND RADIUM THERAPY

ROSSITTO, ANTHONY F. Roentgen treatment of infections of the tonsils and post-pharyngeal lymphoid tissues in children. *Radiology*, February, 1947, *48*, 118–123.

The author treated over 400 children with chronic infections of post-pharyngeal tissues and tonsils referred for roentgen therapy (1) because they had failed to respond to surgical and medical treatment; (2) because of the recurrence of streptococcic infections in the throat after the tonsils and adenoids had been removed; (3) because of persistent unexplained fever following upper respiratory infections, and (4) because they were poor surgical risks.

The author estimates that not less than 90 per cent of the children he treated were cured or showed a good result.

The technical factors used were 200 kv., 0.5 mm. Cu plus 1.0 mm. Al filtration, 50 cm. target-skin distance.

Four to six roentgen treatments given over a period of three to six weeks constituted a course. In rare instances, the number of treatments totaled seven or eight.

The first treatment included the lateral side of the face and sinuses, the pharynx and cervical region. To this field which included the adenoids, pharynx, tonsils and cervical lymph nodes, was delivered 60 to 70 r. Both sides of the head and neck were treated at this time, each of which received the same amount of radiation.

Five to seven days later, 75 r was administered as in the first treatment. In addition, however, 10 to 15 r was added to the exposure in the region of the nasopharynx which received, therefore, a total of 85 to 90 r.

Seven days later, the third treatment was given exactly as it was delivered at the second sitting.

For the fourth treatment, given seven to ten days later, there was an increase in the dosage so that the entire lateral face and cervical area received 85 to 90 r while the nasopharynx was given 95 to 110 r.

The fifth and sixth treatments, if necessary, were given two to three weeks later.

The author is extremely enthusiastic and recommends therapeutic procedure highly to all radiologists.—*Philip J. Hodes, M.D.*

Windholz, Frank. Late changes in mucous membrane of the irradiated larynx; their radiological relationship to the subepithelial connective tissue and to retrogression of laryngeal carcinoma: histologic studies. *Radiology*, March, 1947, *48*, 274–281.

This is the third in a series of four papers. It is recalled that Coutard was the first to describe the early fibrinous reaction to mucous membrane irradiation.

The present study consists of microscopic studies of laryngeal mucous membrane specimens obtained one to ten months after irradiation amounting to an average tissue dose of 5,800 r (Thoraeus filter, 220 kv., 8.1 r per min., field size 6 by 6 cm.).

The degree of fibrinous reaction in the epithelial and connective tissue layers soon after irradiation serves as an index of the future course, with a strong fibrin reaction indicating a favorable result and a weak reaction indicating a poor result. The condition of the subepithelial connective tissue after irradiation determines the structure of the regenerated epithelium. Strong fibrinous reactions produce hyaline sclerotic tissue with collagen in the subepithelial connective tissue layer and an epithelium which is markedly atrophic. Weak fibrin reactions later had well preserved connective tissue and the superficial epithelium showed tendencies to atypical growth with residual tumors.—*Samuel H. Fisher, M.D.*

Lame, Edwin L., and Pendergrass, Eugene P. An addition to the technic of simple breast roentgenography. *Radiology*, March, 1947, *48*, 266–268.

The technical factors of the authors' roentgenography of the breast are: 120 ma., $\frac{1}{2}$ sec., 60 inch target-film distance, 45–55 kv. (peak), screens, 3 inch cone, 0.8 mm. rotating anode.

Initially, several exposures of slightly different factors are obtained to underexpose for the breast periphery and to overexpose for the center of the breast. Subsequent film studies for comparison are made at the same period in the menstrual cycle and a mid-menstrual study is carried out to demonstrate the resting stage.

In studying erect high breasts, the patient stands with the breast in profile; in patients with pendant breasts, the upright films are obtained and, in addition, exposures are made with the patient bending forward and slightly to the side so that the breast hangs free of the thorax. Horizontal positioning of the patient using a vertical central ray spreads the breast flat and thin and is useful for demonstrating axillary nodes.—*Samuel H. Fisher, M.D.*

Palazzo, William L. Lymphoblastoma; an evaluation of the differences in sensitivity to x-ray irradiation of different types, and its application to a quantitative therapeutic test. *Radiology*, May, 1947, *48*, 484–492.

The author attempts to develop a quantitative therapeutic test using roentgen therapy to determine various types of lymphoblastomas based upon their relative radiosensitivity. Arranged in the order of sensitivity, the author considers (*A*) giant follicular lymphadenopathy; (*B*) lymphatic leukemia; (*C*) lymphosarcoma; (*D*) polymorphous-cell sarcoma; (*E*) Hodgkin's disease.

The following routine, as applied to medias-

tinal masses, is suggested. Roentgen rays with the following physical characteristics are employed: 200 kv., 20 ma., 0.5 mm. Cu plus 1.0 mm. Al, target-skin distance 50 cm.

1. A pre-irradiation roentgenogram of the chest allowing measurement of the size of a mediastinal mass is advised.

2. On the first day, 300 r, measured in air, is directed toward the tumor.

3. On the second day, an additional 300 r is directed into the tumor mass.

4. On the fifth day, the roentgenogram of the chest is repeated and the mass compared in size with its original dimensions. If the mass has decreased approximately 25 per cent or more in size, the presumptive diagnosis of either giant follicular lymphadenopathy or lymphosarcoma is made. Therapy is then continued at the discretion of the radiologist in an effort to deliver a tumor-killing dose.

5. If no change is noted in the size of the mass, roentgen therapy is continued at the rate of 200 to 300 r daily, with films of the chest being repeated at three day intervals. If, when 1,200 to 2,000 r has been delivered the mass begins to show regression, a presumptive diagnosis of polymorphous-cell sarcoma is made.

6. If as much as 2,000 to 3,000 r is required to cause a 25 to 50 per cent decrease in the size of the tumor, a presumptive diagnosis of Hodgkin's disease is made.

7. If there is no response with 3,000 r, the tumor is considered radioresistant and not of the lymphosarcoma group.

The author discusses the theoretical possibility of determining differences in cell sensitivity between small cell lymphosarcomas and large cell types, the latter being more radiosensitive. The reticulum cell variety being more the connective tissue type, tends to be somewhat more resistant than the others.—*Philip J. Hodes, M.D.*

Merner, T. B., and Stenstrom, K. W. Roentgen therapy in Hodgkin's disease. *Radiology*, April, 1947, *48*, 355–368.

The authors present a series of 185 cases of Hodgkin's disease, proved by biopsy. The symptoms of Hodgkin's disease are variable depending upon the stage of progression at the time of the patient's first visit. The only complaint may be palpable cervical or axillary lymph nodes. There may be systemic symptoms of greater or lesser degree such as fever, weakness, cough, anorexia, nausea, and vomiting, pruritus, pain due to bone involvement, and loss of weight. Enlarged retroperitoneal nodes are the commonest cause of backache. This symptom is a sufficient indication for roentgen therapy to the retroperitoneal region in the presence of Hodgkin's disease, even in the absence of other positive signs.

The microscopic structure is widely variable in different cases. There are certain features, however, distinctive of Hodgkin's disease, which the author enumerates.

A positive diagnosis of Hodgkin's disease depends entirely upon the histopathologic appearance. However, positive chest roentgen findings, trial roentgen therapy, eosinophilia, pruritus, Pel-Ebstein fever, and lymph node enlargement may point distinctly toward the correct diagnosis. The disease is most common in the third decade. About two-thirds of the patients are males. In America it affects Negroes and white persons to about the same extent.

Since Hodgkin's disease is histopathologically a disease of the reticulo-endothelial system it may be found in almost any organ of the body. The peripheral lymph nodes are the most frequently involved. Bone lesions are most often osteolytic and occur in the ribs, sternum, vertebrae, pelvis, humerus, and femurs. In this series bone lesions, demonstrable by roentgen examination, were present in 20 per cent of the cases.

Irradiation either by roentgen rays or radium is by far the most effective method in the treatment of Hodgkin's disease. This disease is so varied in its manifestations that it is necessary to treat each case individually. Different parts of the body should be treated in different ways, depending upon the accessibility of the involved nodes. Some modification of the treatment is also required in advanced stages. Usually more than one node in a chain is involved, and the whole group should be treated as a unit. For example, if cervical nodes are involved on one side it would seem advisable to include the supraclavicular and submaxillary nodes in a single field. Treatment should also be directed to the mediastinum as a whole, rather than being limited to the area where there is a visible mass. Since recurrences seem more radioresistant, it is advisable to use a heavier dosage during the initial series to prevent their development. The series should be given in a

relatively short time to produce the maximum effect; fourteen days has been arbitrarily chosen as the upper limit. A dose of more than 1,000 tissue roentgens is given in almost all cases, in some cases as much as 2,000 tissue roentgens. More than this amount is rarely used. In the case of cervical nodes, 900 r, in air, is given to each of three fields, an anterior, posterior, and lateral. In the case of the mediastinum, 1,200 to 1,500 r, in air, to each anterior and posterior field is within the proper range of dosage. Very large masses of long standing are usually more resistant to irradiation than smaller, more recently enlarged nodes and should be treated more heavily. The most favorable cases are those in which only one chain of nodes is involved, and thorough irradiation is given after a biopsy has been taken. For the peripheral lesions in this series, 200 to 220 kv. and 0.5 mm. copper plus 1.0 mm. aluminum filter have been used, the half-value layers being 0.9 and 1.3 mm. copper, respectively. For the deeper lesions the filter was increased to 1.0 mm. copper plus 1 mm. aluminum, the half-value layers being 1.4 and 1.7 mm.

In the series reported here the five year survival rate was 21 per cent and the ten year survival rate was 8 per cent, a high figure as compared with other series. The average duration of life from institution of therapy was 33.1 months. Of 5 patients surviving more than ten years from the beginning of treatment, 4 are still alive, the longest period of survival being sixteen years.—*Samuel G. Henderson, M.D.*

Kelby, G. M., and Stenstrom, K. Wilhelm. Treatment of malignant tumors of the testis. *Radiology*, January, 1947, *48*, 1–7.

This study is based on the tumors of the testis treated at the University of Minnesota Hospital from 1926 to 1943 inclusive. The total number amounted to 100 and they were placed in three groups: 34 cases were not classified as to type, while 37 were seminomas and 29 were carcinomatous mixed tumors. True testicular teratomas are rare and the diagnosis cannot be made unless the whole tumor is carefully studied.

The onset of the disease is characteristically insidious. One of the first symptoms noted by the patient is enlargement of the involved testis, and the tumor is often accidentally found. As the disease advances dull pain and discomfort are usually experienced. Backache, a feeling of fullness, and nausea, sometimes with vomiting, are generally associated with metastasis to the retroperitoneal nodes.

Metastases were known to be present in 65 per cent of this series, the most common site being the retroperitoneal nodes. Metastasis to the inguinal region was next in frequency. Metastasis to the retroperitoneal nodes cannot be established until large masses are present. Is is probable that undiagnosed metastatic nodes are present in most patients, particularly in those with highly malignant seminomas. It is for this reason that radiation therapy is given routinely to the retroperitoneal chain of lymph nodes whenever a diagnosis of seminoma has been made following simple orchiectomy. Metastases in the mediastinum, lungs, and superficial nodes can be demonstrated before they have become too large for adequate treatment. As irradiation to all these areas would mean considerable hardship for the patient, the authors have applied roentgen therapy only for proved involvement. A diagnosis of testicular tumor should be made from clinical examination. Biopsy is not advisable, but the testis should be removed for histopathological study if malignant growth is suspected.

The present day treatment of testicular tumors in various clinics consists of simple orchiectomy followed by irradiation. Preoperative irradiation is advocated by some. The plan of treatment followed at the University of Minnesota Hospital up to the present time has consisted of simple orchiectomy and high voltage irradiation in 90 cases, irradiation alone in 6 advanced cases, and Hinman's operation and irradiation in 4 cases. Factors used have been 200 kv. (peak), 15 ma., 1 mm. Cu plus 1 mm. Al filter, half-value layer 1.77 mm. Cu, target skin distance 70 cm. In patients without clinical evidence of metastases at the present time two anterior fields are used, one for the upper abdomen and a lower field which includes the inguinal region of the involved side. A single posterior field extending from the level of the xiphoid to the pubis is used. About 2,000 roentgens in air is given to each field over a period of three weeks. The average daily dose is 200 roentgens in air. If a large abdominal mass is present intense treatment is at first directed to that area only. Later other areas are treated if local response has been obtained and the patient has recovered from the debilitating effect of the earlier therapy. The usual meas-

ures are taken to reduce the irradiation sickness to a minimum.

Results of therapy can be summarized as follows: In seminomas of the testis the five year survival rate is 56 per cent. In the carcinomatous mixed tumor group only one patient survived five years, while in the unclassified group, 29 per cent survived five years. If all the patients with malignant testicular tumors are considered together, a five year survival rate of 29 per cent is obtained.

The authors conclude that simple orchiectomy followed by irradiation is the treatment of choice in seminomas. The carcinomatous mixed tumors should be treated by radical dissection, an adult teratoma by simple orchiectomy.—*Samuel G. Henderson, M.D.*

Windholz, Frank. Problems of acquired radioresistance of cancer: adaptation of tumor cells. *Radiology*, April, 1947, *48*, 398–404.

Acquired radioresistance may be due, according to the summarized opinions of many authors, to:

1. Residual components of radioresistant cells in each tumor producing a radioresistant strain of cells.
2. Production by irradiation of a biologic mutation of radioresistant cancer cells.
3. Increased fibrosis of the tumor bed.
4. Increased maturation of tumor cells under the influence of irradiation.

To this group, the author adds his opinion, based on microscopic examination of the human larynx after irradiation. His observations support the third opinion, mentioned above, that the residual tumor adapts itself to a changed environment of irradiation-induced sclerotic connective tissue in the tumor bed, and acquires increased radioresistance.

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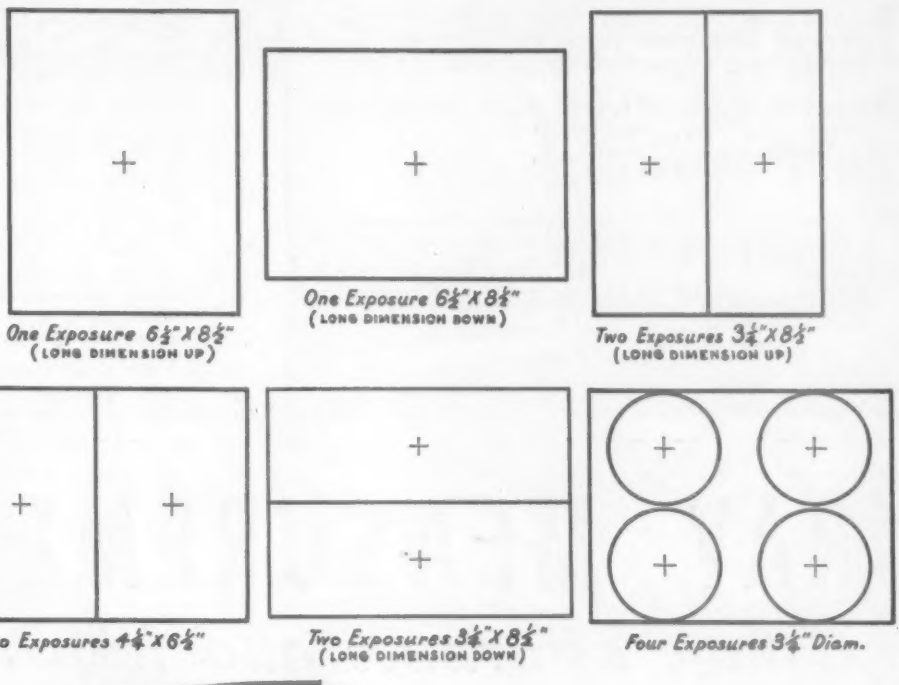

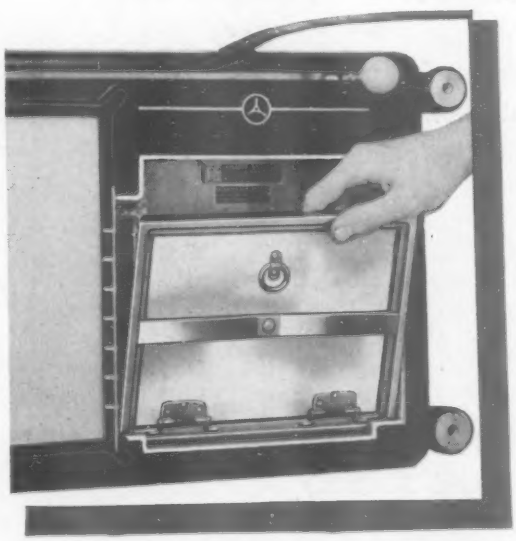

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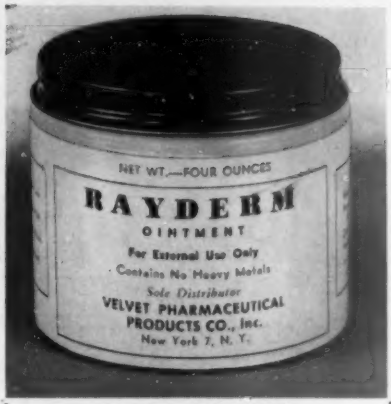

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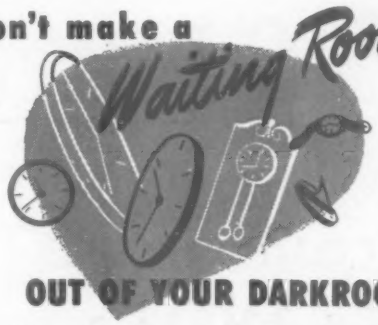

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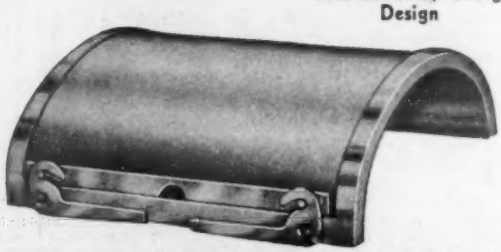

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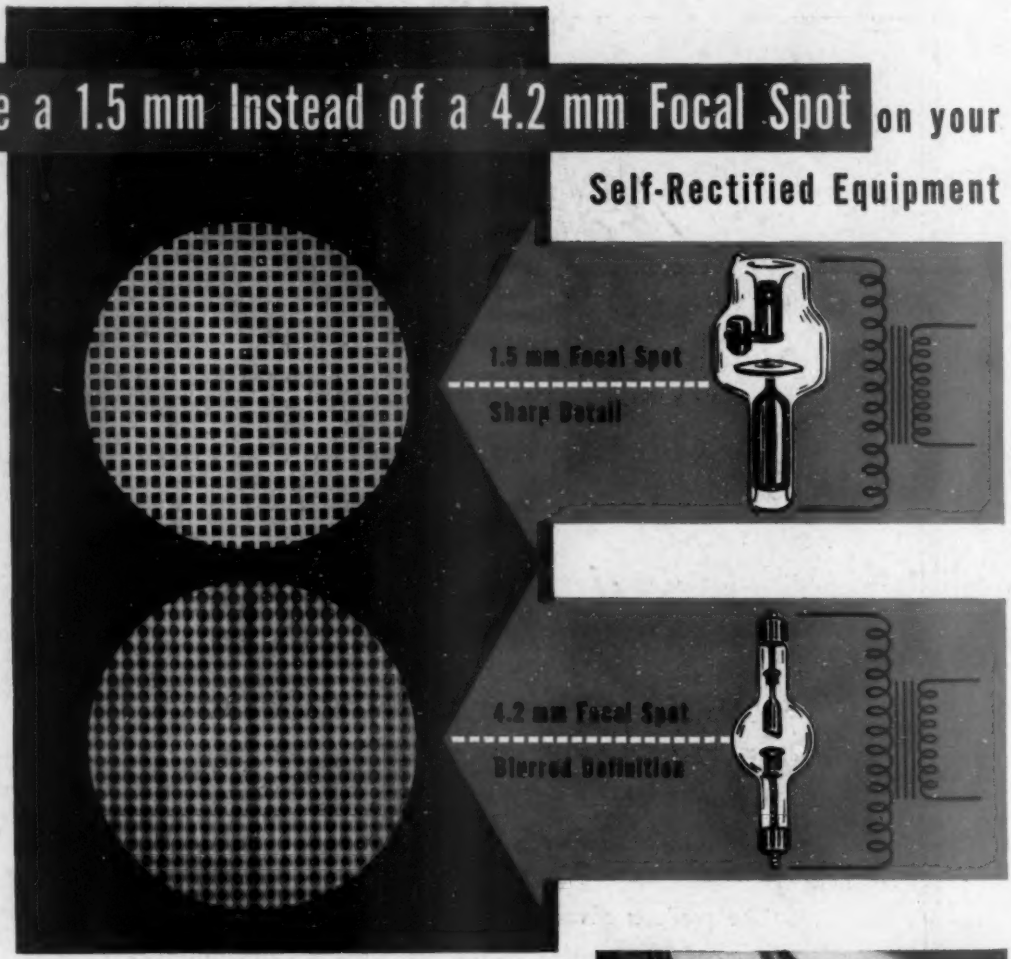

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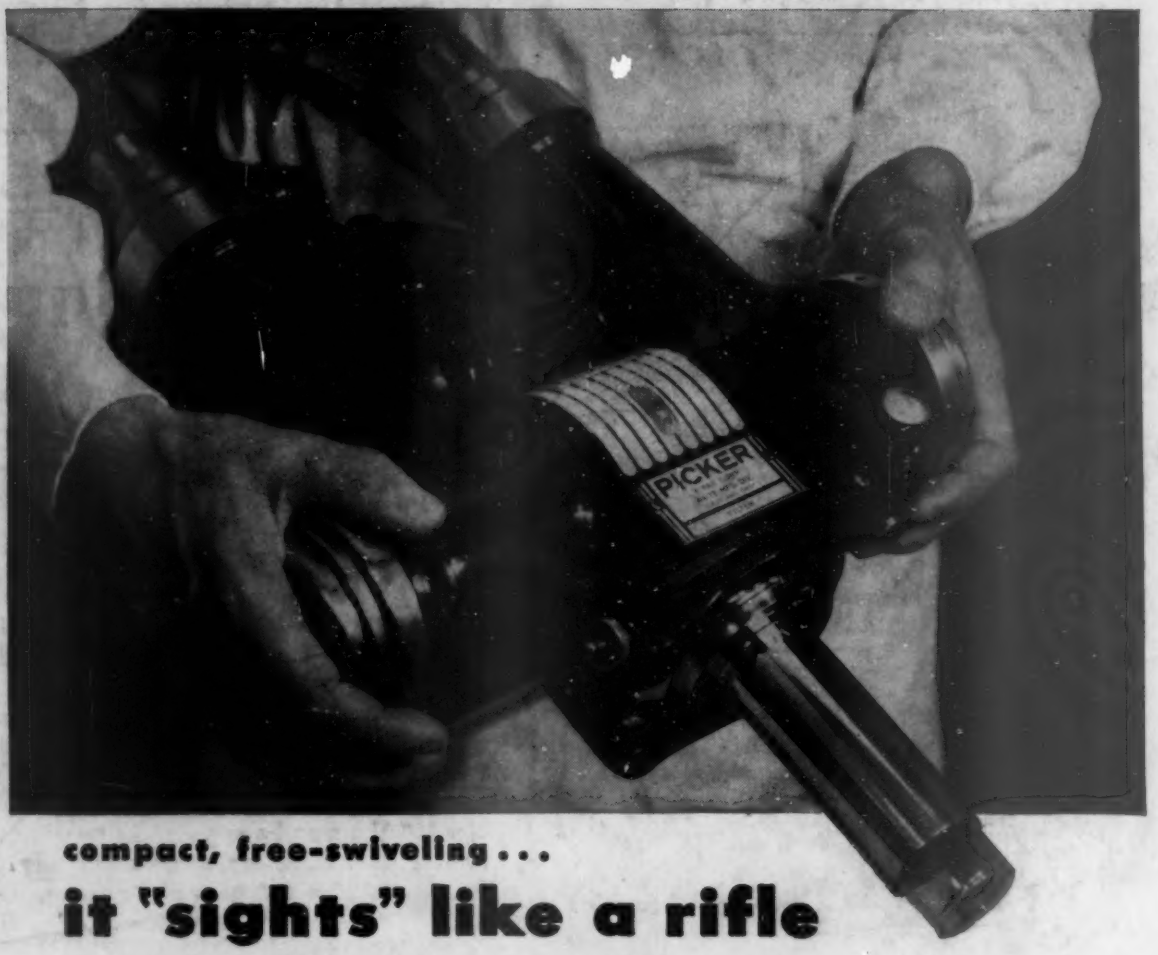

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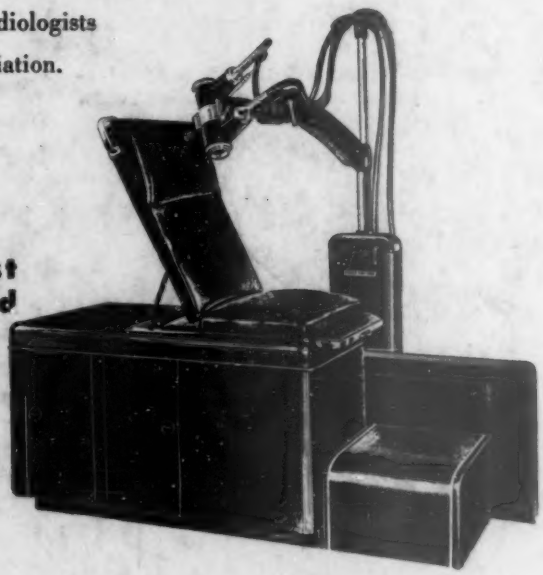

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